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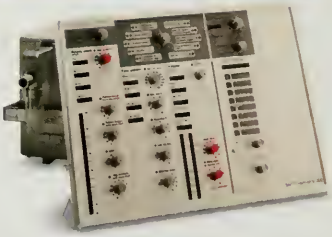

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CONTENTS

February 1994
Volume 39, Number 2

SYMPOSIUM PAPERS

104 New Horizons IX: Cost-Effectiveness of Respiratory Care in the ICU

105 Inhaled Bronchodilators during Mechanical Ventilation: Delivery Techniques, Evaluation of Response, and Cost-Effectiveness
by Dean Hess—Boston, Massachusetts

123 Monitoring Gas Exchange: Clinical Effectiveness and Cost Considerations
by Charles G Durbin Jr—Charlottesville, Virginia

KITTREDGE'S CORNER

138 Flow-Triggering Systems
by Richard D Branson—Cincinnati, Ohio

HISTORICAL NOTES

145 Diseases of the Heart, excerpted from Dr Chase's Third, Last and Complete Receipt Book and Household Physician, by AW Chase MD (published by FB Dickerson Co, Detroit, 1903)
submitted by Crystal L Dunlevy—Columbus, Ohio

BOOKS, FILMS, TAPES, & SOFTWARE REVIEWS

147 Bar Code Technology in Health Care: A Tool for Enhancing Quality, Productivity, and Cost Management, by Karen M Longe and Lisa Brenner
reviewed by John S Sabo—Houston, Texas

147 Dailey's Notes on Blood, 2nd edition, by John F Dailey
reviewed by Shelley Dednan and Mark J Heulitt—Little Rock, Arkansas

147 Pulmonary Diseases and Disorders, 2nd edition, edited by Alfred P Fishman MD
reviewed by C Laura González-Lawless—Salem, Virginia

148 Bronchial Asthma: Mechanisms and Therapeutics, 3rd edition, edited by Earle B Weiss MD and Myron Stein MD
reviewed by Mark W Millard and Gretchen Lawrence—Dallas, Texas

149 Monitoring in Respiratory Care, edited by Robert M Kacmarek PhD RRT, Dean Hess MEd RRT, and James K Stoller MD
reviewed by Rex A Marley—Fort Collins, Colorado

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CONTENTS, *Continued*

February 1994
Volume 39, Number 2

LETTERS

152 Commentary on RCP Smoking Behavior
by D Wayne Martin—Seattle, Washington

153 More on Cigarette Smoking and RCPs
by Dixie Suzor—Toledo, Ohio

153 A "Statistically Significant" Response
by Ellen Becker—Madison, Wisconsin

154 Partial- vs Nonrebreathing Masks
by Emidio M Farias—Montreal, Quebec, Canada

154 Commentary on RETROSPECTROSCOPE
by Leslie Rendell-Baker—Loma Linda, California

ABSTRACTS

86 Summaries of Pertinent Articles from Other Journals

CALL FOR ABSTRACTS

159 Call for OPEN FORUM Abstracts

NOTICES

155 Examination Dates, Notices, Prizes

CALENDAR OF EVENTS

157 Meeting Dates, Locations, Themes

MANUSCRIPT PREPARATION INSTRUCTIONS

161 Instructions for Authors and Typists

NEW PRODUCTS

167 Tuberculosis Drug Reintroduced
167 Oximeter-Hemoglobinometer
167 Emergency Care Organizer
167 Infant CPR Mannequin

INDEXES

168 Authors in This Issue
168 Advertisers in This Issue
101 Advertiser Help Lines

Editorials, Commentaries, and Reviews To Note

Assessing Neonatal Risk: CRIB vs SNAP (commentary)—H Bard. Lancet 1993;342:449.

Improving Cause-of-Death Statistics: The Case of Fetal Deaths (editorial)—D Atkinson. Am J Public Health 1993; 83:1084.

Building Consensus on the Use of Mechanical Ventilation (editorial)—NR MacIntyre. Chest 1993;104:334.

Believe Your Monitors (letter)—JC McAleavy. Anesthesiology 1993;79:409.

Defining the Difficult Airway (letter)—RL Knill. Anesthesiology 1993;79:413.

The Glitter of the *t* Table—D Jolley. Lancet 1993;342:27.

You Cannot Exclude the Explanation You Have Not Considered—M Datta. Lancet 1993;342:345.

Failed or Misleading Adjustment for Confounding—DA Leon. Lancet 1993;342:479.

Accountability, Audit, and Reverence for the Publication Process (editorial)—D Rennie. JAMA 1993;270:495.

Is the Study Worth Doing?—LM Carpenter. Lancet 1993; 342:221.
Bias—C Sitthi-amorn, V Poshyachinda. Lancet 1993;342: 286.

Science Fictions (Editorial Board Speaks)—GW Brown. AJDC 1993;147:809.

Estimating the Effects of Misclassification—TE Mertens. Lancet 1993;342:418.

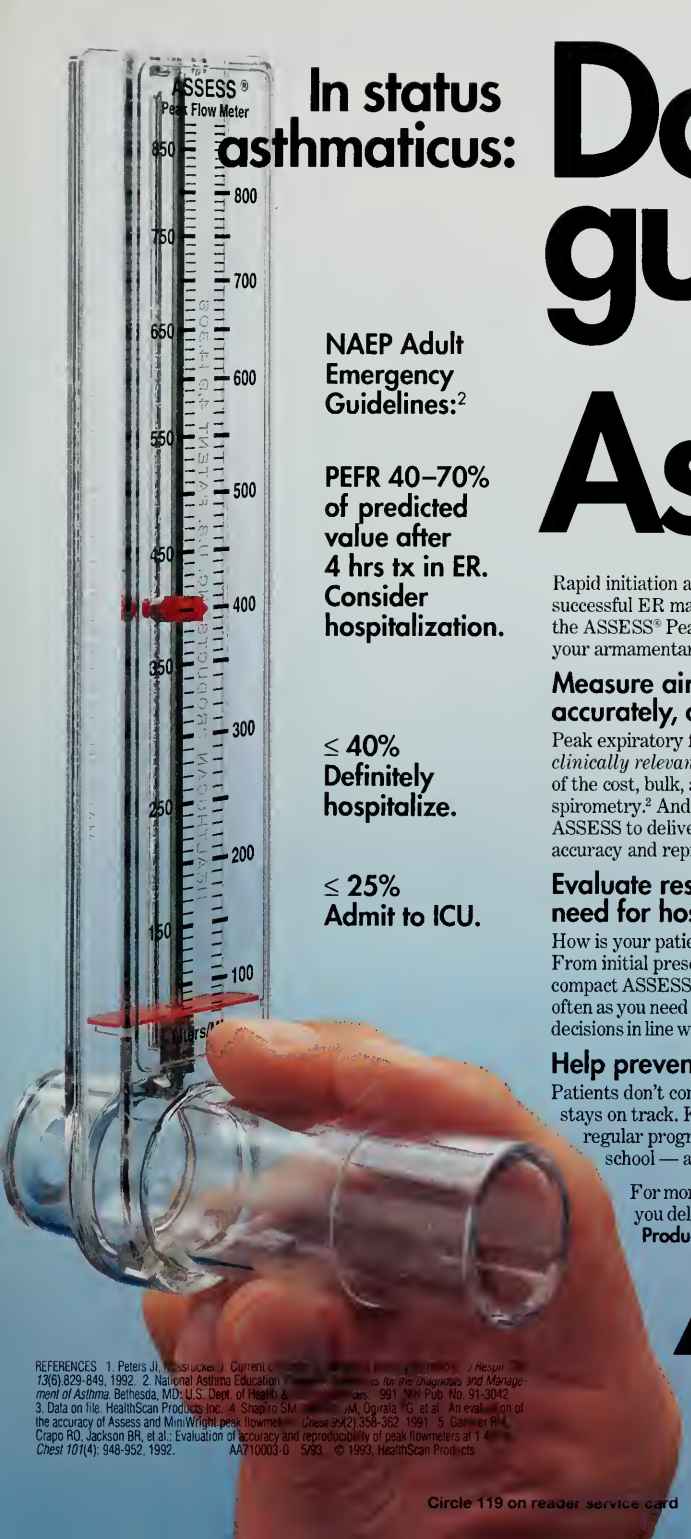
Intracorporeal Respiratory Support: A Potential Supplement to Airway Pressure Therapy? (editorial)—BA Shapiro, WT Peruzzi. Chest 1993;103:1-2. (Pertains to Conrad et al paper abstracted on Page 95.)

Adults with Tetralogy of Fallot—Repaired, Yes; Cured, No (editorial)—A Rosenthal. N Engl J Med 1993;329:655. (Pertains to Murphy et al paper abstracted on Page 102.)

A Question of Attribution—JR Glynn. Lancet 1993;342: 530.

The Effect of Sensor Malpositioning on Pulse Oximeter Accuracy during Hypoxemia—SJ Barker, J Hyatt, NK Shah, J Kao. Anesthesiology 1993;79(2):248.

BACKGROUND: Previous studies have shown that pulse oximeters whose sensors are positioned improperly may yield erroneously low saturation (S_{pO_2}) values on normoxemic subjects. The behavior of oximeters with malpositioned sensors during hypoxemia has not been studied. The current study is aimed at determining the behavior of several different pulse oximeters over a wide range of arterial oxygen saturation (S_{aO_2}). METHODS: In each of 12 healthy volunteers, a radial artery cannula was inserted, and eight different pulse oximeters, five of which had malpositioned sensors, were applied. Subjects breathed controlled mixtures of nitrogen and oxygen to slowly vary their S_{aO_2} from 100% to 70%. Arterial blood samples were analyzed and pulse oximeter data were recorded at five stable S_{aO_2} values for each subject. RESULTS: The oximeters with malpositioned sensors vary greatly in their behavior, depending on both the actual S_{aO_2} and the manufacturer and model. One oximeter underestimated saturation at all S_{aO_2} values, while three others underestimated at high S_{aO_2} and overestimated at low S_{aO_2}. Linear regression analysis shows a decrease in the slope of S_{pO_2} versus S_{aO_2} in most cases, indicating a loss of sensitivity to S_{aO_2} changes. Between-subject variation in response curves was significant. CONCLUSIONS: The calibration curves of the pulse oximeters studied were changed greatly by sensor malpositioning. At low S_{aO_2} values, these changes could



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cause the oximeter to indicate that a patient was only mildly hypoxemic when, in fact, hypoxemia was profound. It is recommended that sensor position be checked frequently and that inaccessible sensor locations be avoided whenever possible.

Use of Capnography for Assessment of the Adequacy of Alveolar Ventilation during Weaning from Mechanical Ventilation—TF Morley, J Giaimo, E Maroszan, J Bermingham, R Gordon, R Griesback, et al. Am Rev Respir Dis 1993;148: 339.

A prospective study was conducted to determine the reliability of noninvasive end-tidal CO_2 (P_{etCO_2}) monitoring as a reflection of arterial CO_2 tension (P_{aCO_2}) during weaning from mechanical ventilation (MV). Simultaneous P_{aCO_2} and P_{etCO_2} determinations were compared during MV and again during a spontaneous breathing trial just before returning the patient to MV. Three groups of patients recovering from acute respiratory failure were evaluated. Group 1 consisted of 16 patients (28 observations) without parenchymal lung disease. Group 2 consisted of 22 patients (31 observations) with alveolar filling diseases. Group 3 was composed of 13 patients (22 observations) with emphysema. Significant Pearson correlation coefficients were demonstrated between P_{aCO_2} and P_{etCO_2} during both MV and spontaneous breathing in all three groups. Significant correlation was also demonstrated between the change in P_{aCO_2} and the change in P_{etCO_2} associated with weaning for each group; however, the degree of correlation varied between groups. Our data suggest that capnography offers a reasonable estimate of P_{aCO_2} and changes in P_{aCO_2} during weaning in patients without parenchymal lung disease. However, P_{etCO_2} is less sensitive to changes in P_{aCO_2} for patients with parenchymal lung disease, particularly patients with emphysema. Interpretation of capnographic data requires a full understanding of its limitations. An approach to capnographic monitoring during weaning is discussed.

The Esophageal Detector Device: Does It Work?—L Zaleski, D Abello, MJ Gold. Anesthesiology 1993; 79(2):244.

BACKGROUND: The esophageal detector device (EDD) is a diagnostic tool for confirmation of tracheal intubation. Capnography is the accepted standard for such confirmation. The purpose of this investigation was to determine whether results using the EDD and capnography agree. METHODS: Five hundred patients were divided into three separate studies. In Study 1, with 300 consecutive patients, tracheal intubation was performed and tested with the EDD followed by capnography. In Study 2, 100 patients had the esophagus intentionally intubated, and confirmation was tested similarly. The tube was then removed and the trachea intubated, and testing followed. Study 3 involved 100 patients and used a double-blind, randomized design. The tube was intentionally inserted into either the esophagus (n = 51) or trachea (n = 49), and testing followed. RESULTS: In Study 1, the compressed EDD bulb reinflated 270 times and always agreed with capnography; in 20 of the 270 subjects (7%) bulb reinflation was delayed, taking from 5-30 s. In 30 instances, the bulb remained compressed, and there was no capnogram indicating esophageal intubation. In Study 2, regardless of esophageal or tracheal intubation, agreement between EDD and capnogram was 100%. In Study 3, the agreement between the two detecting instruments was 100%, but reinflation of the EDD bulb was delayed in 4% of tracheal intubations. In the 500 patients studied, results from the EDD and capnogram always agreed, but in 6% of all tracheal intubations, the EDD bulb inflated slowly. Of 181 esophageal intubations, the results from the EDD and capnogram always agreed (ie, there was no reinflation or capnogram). The sensitivity, specificity, and predictive value for the EDD in all of these studies was 100%. CONCLUSIONS: The EDD is a valuable diagnostic technique for confirming tracheal intubation. Results using EDD agree with results using capnography; in 6% of instances there is a slow reinflation; and where there is no capnography, such as on hospital wards, EDD may be a useful diagnostic tool.

Pitfalls in the Care of Patients with Tuberculosis: Common Errors and their Association with the Acquisition of Drug Resistance—A Mahmoudi, MD Iseman. JAMA 1993;270: 65.

OBJECTIVE: To determine, among a group of patients with multidrug-resistant pulmonary tuberculosis, whether there had been management practices that deviated from established guidelines, and whether these decisions were associated with the acquisition of multidrug resistance and adverse medical sequelae. DESIGN: Case series. SETTING: Referral center. PATIENTS: All patients with pulmonary tuberculosis admitted to the National Jewish Center for Immunology and Respiratory Medicine in 1989 through 1990. INTERVENTIONS: The records of all patients referred to this institution for the treatment of tuberculosis in 1989 through 1990 were reviewed to ascertain the nature of management decisions that might have been associated with the acquisition of drug resistance. MAIN OUTCOME MEASURES: Standards of practice as de-

fined by the American Thoracic Society, the Centers for Disease Control and Prevention, and the American College of Chest Physicians were compared with these management decisions to determine whether "errors" had been made, resulting in treatment failure and the development of acquired drug resistance. RESULTS: Among the 35 study patients, errors were detected in the management decisions in 28; there was an average of 3.93 errors per patient. The most common errors were the addition of a single drug to a failing regimen, failure to identify preexisting or acquired drug resistance, initiation of an inadequate primary regimen, failure to identify and address noncompliance, and inappropriate isoniazid preventive therapy. The multidrug resistance acquired through the errors resulted in prolonged hospitalizations, treatment with more toxic drugs, and high-risk resectional surgery. The costs for this "salvage therapy" were extraordinary, averaging $180,000 per patient. CONCLUSIONS: Aggressive professional education, tighter control on the provisions of care for tuberculosis patients, and the committing of additional resources to tuberculosis control programs are vital in improving the care of tuberculosis patients and limiting the development of acquired drug resistance.

Comparison of Blue Dye Visualization and Glucose Oxidase Test Strip Methods for Detecting Pulmonary Aspiration of Enteral Feedings in Intubated Adults—RG Potts, MH Zaroukian, PA Guerrero, CD Baker. Chest 1993;103:117.

STUDY OBJECTIVE: To compare the relative utility of blue dye visualization with a glucose oxidase test strip method for detecting aspiration of enteral feedings. DESIGN: Tracheally intubated adults were prospectively monitored for aspiration of enteral feedings. SETTING: Intensive care units of two community hospitals in Michigan. INTERVENTIONS: None. PATIENTS: The experimental group consisted of 15 patients receiving enteral feedings. The control group included 14 patients not enterally fed. MEASUREMENTS & RESULTS: Blue food coloring was added to feeding formulas to obtain a visible blue color. At 8-h intervals, tracheal secretions were examined for blue discoloration, followed by measurement of glucose concentration using a calibrated glucose meter. Clinically significant aspiration was defined to require the following: (1) a bloodless positive glucose reading (≥ 20 mg/dL; (2) one or more signs of systemic inflammation; and (3) one or more signs of respiratory deterioration. Eight (53%) of 15 patients in the experimental group experienced at least one episode of presumptive aspiration as defined by either a bloodless positive glucose reading or visible blue discoloration of tracheal secre-

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tions. Clinically significant aspiration occurred in 5 (33%) of 15 patients in whom bloodless glucose readings were positive in 13 (19%) of 67 samples; among patients not developing this complication, glucose was found in only 3 (5%) of 60 samples; ($p = 0.005$). Inspecting tracheal secretions for blue dye usually failed to detect aspiration episodes identifiable by the glucose oxidase test strip method (relative sensitivity, 13%). Blue dye visualization performed no better among patients developing clinically significant aspiration (relative sensitivity, 15%). Patients who developed clinically significant aspiration received more of their enteral feedings in the supine position than patients without this complication (98% vs 21%; $p < 0.001$). CONCLUSIONS: Inspecting tracheal secretions for blue discoloration failed to detect most episodes of enteral feeding aspiration. Glucose oxidase test strip methods should replace blue dye visualization for detecting aspiration of enteral feedings in intubated adults.

Prolonged Intracorporeal Support of Gas Exchange with an Intravenacaval Oxygenator—SA Conrad, JM Eggerstedt, VF Morris, MD Romero. Chest 1993;103:158.

Patients with severe respiratory failure undergoing conventional mechanical ventilation typically are exposed to levels of ventilator support that place the patient at risk of barotrauma or oxygen toxicity. In severe cases, gas exchange may be inadequate despite maximal ventilator support. We report two cases of advanced respiratory failure in which augmentation of gas exchange was provided for prolonged periods (18 and 13 days) with an intravenacaval hollow-fiber membrane oxygenator (IVOX). Following implantation, significant reductions in ventilator support were possible with improvement in arterial blood gas values. No significant complications were noted. IVOX can provide clinically useful augmentation of gas exchange in respiratory failure and can be used for prolonged periods.

Continuous Positive Airway Pressure in COPD Patients in Acute Hypercapnic Respiratory Failure—AM Miro, U Shivaram, I Hertig. Chest 1993;103:266.

We used mask continuous positive airway pressure (CPAP) in seven patients with acute hypercapnic respiratory failure in an attempt to avoid endotracheal intubation and mechanical ventilation. Mask CPAP was started at 5 cm H_2O and then increased to a maximum of 10 cm H_2O depending on the clinical response. In five patients, CPAP significantly improved gas exchange; four of these patients were weaned to oxygen by face mask without needing intubation. In two patients, gas exchange deteriorated even with CPAP of 10 cm H_2O. No barotrauma or adverse hemodynamic effects were associated with CPAP. We conclude that a trial of mask CPAP may be warranted before intubation of an alert acutely hypercapnic patient with COPD.

Intermittent Positive Pressure Ventilation via the Mouth as an Alternative to Tracheostomy for 257 Ventilator Users—JR Bach, AS Alba, LR Saporito. Chest 1993;103: 174.

Despite wider application of the use of nocturnal intermittent positive pressure ventilation (IPPV) via nasal access for the management of nocturnal hypoventilation, there continues to be a lack of familiarity with the use of IPPV via the mouth for ventilatory support. Unlike nasal IPPV, which is generally practical only for nocturnal use, up to 24-h mouth IPPV was the key method of noninvasive ventilatory support that permitted the avoidance or elimination of tracheostomy for 257 individuals with acute or chronic ventilatory failure. Mouth IPPV was delivered via commercially available mouthpieces for daytime aid and mouthpiece with lip seal or custom orthodontic interfaces for nocturnal support. The use of mouth IPPV alone or in a regimen with other noninvasive ventilatory aids was reviewed for these 257 individuals. Mouth IPPV was used for nocturnal aid by 163 individuals, 61 of whom had little or no measurable vital capacity or significant ventilator-free breathing time, for more than 1,560 patient-years with few complications. It was also the predominant method of daytime ventilatory support for 228 individuals for more than 2,350 patient-years. We conclude that for individuals with adequate bulbar muscle function but chronic respiratory muscle insufficiency, mouth IPPV can be an effective alternative to tracheostomy. It can significantly prolong survival while optimizing convenience, safety, and communication.

Chest Radiograph—A Poor Method for Determining the Size of a Pneumothorax—O Engdahl, T Toft, J Boe. Chest 1993;103:26.

The ability of chest radiographs to determine the size of a pneumothorax was tested in 16 patients using computed tomographic (CT) scan as a reference method. To determine if CT with a slice thickness of 12 mm could be used, its accuracy was assessed in a lung model experiment. The lung model consisted of a water-filled plastic bag (lung) fitted into a plastic chamber (hemithorax), both of approximately the same size and shape as in man. Water was drawn off in incremental steps and a CT was done after each step. The area of

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the pneumothorax was calculated by computer and when multiplied by slice thickness and number of slices, the total volume of the pneumothorax good correlation (r = 0.99), with the line of regression close to the line of identity, was found between the CT investigation and the artificial pneumothorax. In the patients, the size of the pneumothorax, judged by radiograph using two different methods of calculation, was correlated to the size obtained by CT. The correlation was poor (r = 0.71) irrespective of method of calculation. The size of the pneumothorax estimated by CT showed a good correlation (r = 0.99) to the initial aspirated air volumes in 12 of the 16 patients treated with drainage. A cautious attitude toward the use of chest radiographs for calculations of the degree of lung collapse in patients with pneumothorax is recommended.

Pulmonary Rehabilitation That Includes Arm Exercise Reduces Metabolic and Ventilatory Requirements for Simple Arm Elevation—JI Couser Jr, FJ Martinez, BR Celli. Chest 1993;103:37.

Simple arm elevation results in increased metabolic and ventilatory requirements in patients with chronic airflow obstruction (CAO). These demands contribute to the dyspnea that is frequently reported when these patients perform activities of daily living involving the arms. We hypothesized that a comprehensive pulmonary rehabilitation (PR) program that includes upper extremity training would lower the ventilatory requirement for arm elevation. Metabolic and ventilatory responses to 2 min of simple arm elevation were studied in 14 patients with CAO before and after PR. Respiratory muscle strength was determined in 11 patients by measurement of maximal transdiaphragmatic pressure (P_{dimax}). Oxygen uptake ($\dot{V}_{O_2}$), carbon dioxide production ($\dot{V}_{CO_2}$), heart rate (HR), minute ventilation (V_E), tidal volume (V_T), and respiratory rate were measured at rest with the arms down and during 2 min of arm elevation. Before PR, arm elevation led to significant increases in $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, HR, and V_E. After PR, pulmonary function, P_{dimax}, and resting metabolic and ventilatory parameters with the arms down were unchanged; however, during arm elevation, $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, and V_E were significantly less than they were before PR. We conclude that a comprehensive PR program that includes upper extremity exercises leads to a reduction in the ventilatory requirement for simple arm elevation. This type of program may allow patients with CAO to perform sustained upper extremity activities with less dyspnea.

Maximal Inspiratory Pressure: Learning Effect and Test-Retest Reliability in Patients with Chronic Obstructive Pulmonary Disease—JL Larson, MK Covey, CA Vitalo, CG Alex, M Patel, MJ Kim. Chest 1993;104:448.

Reliability of the maximal inspiratory pressure ($P_{I_{max}}$) was examined by measuring $P_{I_{max}}$ once a week for 4 weeks in 91 patients with chronic obstructive pulmonary disease using an aneroid pressure gauge. Five $P_{I_{max}}$ trials were conducted at each test. From the first to the fourth test, the $P_{I_{max}}$ increased by a mean of 9 cm H_2O (SD = 10). From the third to the fourth test, $P_{I_{max}}$ increased by a mean of 2 cm H_2O and performance appeared to be plateauing. The test-retest reliability coefficient was r = 0.97 for $P_{I_{max}}$ measured at the third and fourth test session. The 95 percent confidence interval for the absolute difference in $P_{I_{max}}$ at the third and fourth test was 3 to 5 cm H_2O. We conclude that performance of $P_{I_{max}}$ improves with practice in naive COPD patients and $P_{I_{max}}$ is reliable when measured with an aneroid gauge by experienced data collectors if patients are given sufficient practice.

The Long-Term Benefits of Outpatient Pulmonary Rehabilitation on Exercise Endurance and Quality of Life—F Vale, JZ Reardon, RL ZuWallack. Chest 1993;103:42.

Although it is generally accepted that outpatient pulmonary rehabilitation (OPR) improves exercise performance and quality of life (QOL), it is not well established whether these gains are sustained over time. To evaluate this, we attempted to contact the 71 patients who had completed our 6-week OPR program for follow-up 12-min walking distance (12 MD) and QOL measurements. Fifty-one patients (71.8%) returned for follow-up testing 11.0 ± 6.1 months following OPR. Of these, 19 had participated in a structured post-OPR exercise maintenance (EM) program, while 32 had not (non-EM). The 12 MD increased from 2,300 ± 611 ft at baseline to 2,789 ± 622 ft post-OPR, while the QOL (higher is better) increased from 81.2 ± 21.4 to 104.7 ± 22.2 over this period (both, $p < 0.0001$). Although the 12 MD decreased by 10.6 ± 15.8% to 2,539 ± 803 ft at follow-up ($p < 0.001$), it remained 10.3 ± 23.4% greater than baseline ($p < 0.001$). Similarly, despite decreasing by 7.6 ± 13.6% to 96.4 ± 23.3 ($p < 0.005$), the follow-up QOL remained 22.8 ± 35.9% greater than baseline ($p < 0.005$). The post-OPR to follow-up declines in 12 MD and QOL were not significantly different between EM and non-EM patients. Thus, only a portion of the initial improvement in exercise endurance and QOL is lost at follow-up months later. Post-OPR EM did not appear to provide measurable long-term advantages.

Racial Differences in the Use of Invasive Cardiovascular Procedures in the Department of Veterans Affairs Medical System—J Whittle, J Conigliaro, CB Good, RP Lofgren. N Engl J Med 1993;329:621.

BACKGROUND: Previous studies have found racial differences in the use of invasive cardiovascular procedures, which may be due in part to the greater financial incentives to perform such procedures in White patients. In Department of Veterans Affairs hospitals, direct financial incentives affecting use of the procedures are minimized for both patients and physicians. METHODS: We analyzed retrospectively the use of cardiovascular procedures among Black and White male veterans discharged from Veterans Affairs hospitals with primary diagnoses of cardiovascular disease or chest pain during fiscal years 1987 through 1991. We used coded discharge data to determine whether cardiac catheterization, percutaneous transluminal coronary angioplasty, or coronary artery bypass grafting was performed during or immediately after such admissions. We used logistic-regression analysis to adjust for the primary discharge diagnosis, the presence of coexisting conditions, age, marital status, type of eligibility to receive care at Veterans Affairs hospitals, geographic region, and whether the hospital was equipped to perform bypass surgery. We classified the primary diagnosis as myocardial infarction, unstable angina, angina, chronic ischemia, chest pain, or "other" cardiovascular diagnosis. RESULTS: After we adjusted for all the potential confounders, we found that White veterans were more likely than Black veterans to undergo cardiac catheterization (odds ratio, 1.38; 95% confidence interval, 1.34 to 1.42), angioplasty (odds ratio, 1.50; 95% confidence interval, 1.38 to 1.64), and coronary artery bypass surgery (odds ratio, 2.22; 95% confidence interval, 2.09 to 2.36). CONCLUSIONS: Even when financial incentives are absent, Whites are more likely than Blacks to undergo invasive cardiac procedures. These findings suggest that social or clinical factors affect the use of these procedures differently in Blacks and Whites.

Nasal Continuous Positive Airway Pressure in Decompensated Hypercapnic Respiratory Failure as a Complication of Sleep Apnea—U Shivaram, ME Cash, A Beal. Chest 1993;104:770.

Cardiopulmonary failure resulting from progression of obstructive sleep apnea (OSA) is treated with endotracheal intubation and mechanical ventilation. This study was conducted to determine whether the use of nasal continuous positive airway pressure (CPAP) would rapidly reverse changes in mental status and hypercapnic acidosis in such patients with decompensated hypercapnic respiratory failure resulting from OSA. Six morbidly obese patients (mean weight, 159 ± 19 kg) were treated with nasal CPAP and supplemental oxygen. Within 24 h of this treatment, there was a significant increase in pH, from a baseline mean of 7.23 ± 0.03 to 7.35 ± 0.01 ($p < 0.01$), and the mean P_{aCO_2} fell from 80 ± 4 torr to 64 ± 4 torr ($p < 0.01$). In addition, there was a dramatic improvement in mental status within 24 h of therapy with nasal CPAP. None of the patients required intubation and mechanical ventilation. There were no complications attributable to the CPAP delivered by nasal mask. We conclude that CPAP delivered by nasal mask can be safe and effective in rapidly reversing changes in mental status and hypercapnic acidosis in this group of patients with severe respiratory failure, and nasal CPAP obviates the need for endotracheal intubation and mechanical ventilation.

Mechanical Ventilation for *Pneumocystis carinii* Pneumonia in Patients with the Acquired Immunodeficiency Syndrome: Is the Prognosis Really Improved?—F Staikowsky, B Lafon, B Guidet, M Denis, C Mayaud, G Offenstadt. Chest 1993;104:756.

The mortality rate among patients with human immunodeficiency virus (HIV) requiring mechanical ventilation (MV) for acute respiratory failure (ARF) secondary to *Pneumocystis carinii* pneumonia (PCP) is still a matter of discussion. For some authors, it is in the 50% range, while for others the prognosis is grim, with virtually no survivors. The aim of this retrospective study conducted between January 1987 and January 1992 was to analyze the outcome of such patients. We studied 33 patients, 29 men and 4 women (38.6 ± 9.9 years, 21 homosexuals, 8 intravenous drug users, 3 transfusion related, 1 heterosexual) infected by HIV for at least 19.7 ± 21.6 months. It was the first PCP episode in all but 2 patients and the diagnosis was made by bronchoalveolar lavage (n = 32) or lung biopsy specimen (n = 1). Only three patients were receiving primary prophylaxis for PCP (trimethoprim-sulfamethoxazole [TMP-SMZ], n = 2; pentamidine, n = 1). *Pneumocystis carinii* pneumonia was the first manifestation of AIDS in 9 patients. The duration of symptoms prior to treatment was 19.6 ± 11.3 days. At the time of hospital admission, laboratory findings were as follows: P_{aO_2} = 40.7 ± 7.8 torr on room air; serum LDH = 1,172 ± 792 IU/L; T^4 cell count = 60.2 ± 67/mm^3. Mechanical ventilation was always required for ARF, which was never induced by bronchoscopy. The interval between treatment and MV was 8.1 ± 6.5 days and the duration of MV

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THE FDA MEDICAL PRODUCTS REPORTING PROGRAM

For VOLUNTARY reporting by health professionals of adverse events and product problems

Form Approved. OMB No. 0910-0291 Expires 12/31/94
See OMB statement on reverse

FDA Use Only (RESP CARE)

Triage unit sequence #

Page ___ of ___

A. Patient information

1. Patient identifier	2. Age at time of event: or Date of birth:	3. Sex	4. Weight
In confidence		☐ female ☐ male	___ lbs or ___ kgs

B. Adverse event or product problem

1. ☐ **Adverse event** and/or ☐ **Product problem** (e.g., defects/malfunctions)

2. **Outcomes attributed to adverse event** (check all that apply)
   - ☐ death (mo/day/yr)
   - ☐ life-threatening
   - ☐ hospitalization – initial or prolonged
   - ☐ disability
   - ☐ congenital anomaly
   - ☐ required intervention to prevent permanent impairment/damage
   - ☐ other:

3. Date of event (mo/day/yr)	4. Date of this report (mo/day/yr)

5. **Describe event or problem**

6. **Relevant tests/laboratory data,** including dates

7. **Other relevant history, including preexisting medical conditions** (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1. **Name** (give labeled strength & mfr/labeler, if known)
   #1
   #2

2. Dose, frequency & route used	3. Therapy dates (if unknown, give duration) from/to (or best estimate)
#1	#1
#2	#2

4. **Diagnosis for use** (indication)
   #1
   #2

5. **Event abated after use stopped or dose reduced**
   #1 ☐ yes ☐ no ☐ doesn't apply
   #2 ☐ yes ☐ no ☐ doesn't apply

6. Lot # (if known)	7. Exp. date (if known)
#1	#1
#2	#2

8. **Event reappeared after reintroduction**
   #1 ☐ yes ☐ no ☐ doesn't apply
   #2 ☐ yes ☐ no ☐ doesn't apply

9. **NDC #** (for product problems only)
   – –

10. **Concomitant medical products** and therapy dates (exclude treatment of event)

D. Suspect medical device

1. **Brand name**

2. **Type of device**

3. **Manufacturer name & address**

4. **Operator of device**
   - ☐ health professional
   - ☐ lay user/patient
   - ☐ other:

5. **Expiration date** (mo/day/yr)

6.
   model # ___
   catalog # ___
   serial # ___
   lot # ___
   other #

7. **If implanted, give date** (mo/day/yr)

8. **If explanted, give date** (mo/day/yr)

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   ☐ yes ☐ no ☐ returned to manufacturer on ___ (mo/day/yr)

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1. **Name, address & phone #**

2. Health professional?	3. Occupation	4. Also reported to
☐ yes ☐ no		☐ manufacturer ☐ user facility ☐ distributor

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- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
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Report even if:

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Report product problems – quality, performance or safety concerns such as:

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- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

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was 11.4 ± 9.9 days. The patients were classified into 3 groups on the basis of the duration and type of treatment before MV, as follows: Group 1, n = 10: TMP-SMZ (20-100 mg/kg) I.V. and methylprednisolone (MP) < 5 days before MV; Group 2, n = 4: TMP-SMZ ≥ 5 days and MP < 5 days; Group 3, n = 19: TMP-SMZ and MP ≥ 5 days before MV. (The MP dose was as follows: 240 mg/d once a day from Day 1 to Day 3; 120 mg/d from Day 4 to Day 6; and 60 mg/d from Day 7 to Day 9.) Despite MV, TMP-SMZ, and MP, death secondary to PCP-related ARF occurred in 81.9% of patients, 20 ± 4.8 days after the beginning of treatment and 11.4 ± 9.9 days after the beginning of MV. Six patients survived, five in Group 1 and one in Group 3. No prognostic factors were found to identify survivors from nonsurvivors, apart from the interval between the beginning of medical treatment and MV (2.8 ± 5.5 vs 9.3 ± 6.1 days, p 0.05) and the duration of known seropositivity (3.2 ± 7.3 vs 23.4 ± 22.1 months, p 0.05). In conclusion, the mortality rate was 50% among the patients in whom MV was started early in the course of PCP-related ARF, but reached almost 100% when MV was started after 5 days of treatment with TMP-SMZ and high-dose I.V. steroids.

Treatment of Orthostatic Hypotension with Erythropoietin—RD Hoeldtke, DHP Streeten. N Engl J Med 1993;329:611.

BACKGROUND & METHODS: Patients with orthostatic hypotension caused by autonomic neuropathy frequently have a decreased red-cell mass. This would be expected to compromise their effective circulating blood volume and aggravate the orthostatic hypotension. We studied the effect of increasing the red-cell mass with erythropoietin, given subcutaneously in a dose of 50 U per kilogram of body weight three times a week for 6 to 10 weeks to eight patients with orthostatic hypotension—four men, one teenage boy, and three women (age range, 17 to 68 years). Four patients had Type I diabetes mellitus and autonomic neuropathy, three patients had pure autonomic failure, and one patient had sympathotonic orthostatic hypotension. Seven patients received fludrocortisone (0.1 or 0.2 mg per day) before, during, and after the trial of erythropoietin. The red-cell volume, plasma volume, and hemodynamic response to orthostatic stress were measured before and after therapy. RESULTS: Erythropoietin increased the mean (± SD) hematocrit from 0.34 ± 0.04 to 0.45 ± 0.04 ($p < 0.005$) and increased the red-cell volume from 16.8 ± 3.9 to 25.3 ± 3.1 mL per kilogram ($p < 0.005$), but had no effect on plasma volume. The systolic blood pressure increased from 81 ± 11 to 100 ± 24 mm Hg ($p < 0.01$) and the diastolic blood pressure increased from 46 ± 10 to 63 ± 18 mm Hg ($p < 0.01$) while the patients were standing. The average systolic and diastolic blood pressure while the patients were supine did not increase significantly, although hypertension in the supine position developed in three patients. Orthostatic dizziness improved during treatment in six of the eight patients. CONCLUSIONS: In patients with orthostatic hypotension, increasing the red-cell volume with erythropoietin elevates blood pressure while standing. Possible long-term adverse effects are not known.

Tobacco Use: A Modifiable Risk Factor for Dental Disease among the Elderly—AM Jette, HA Feldman, SL Tennstedt. Am J Public Health 1993;83:1271.

OBJECTIVES: Because the public health literature contains few analytic studies of modifiable behavioral risk factors for dental disease among older community-dwelling populations, the New England Elders Dental Study was undertaken as an epidemiologic study of the oral health status of a representative sample of older adults living within the six New England states. METHODS: Five dentists conducted comprehensive in-home oral health examinations on 1,156 community-dwelling adults aged 70 and older to determine whether lifetime use of tobacco products was a significant risk factor for tooth loss, caries, and periodontal disease. RESULTS: Among New England elders, tobacco use was more common among men (18.1%) than women (7.9%), with a combined rate of 12.3%. Further, 64.7% of men and 36.6% of women were previous tobacco users. Years of exposure to tobacco products was

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a statistically significant risk factor for tooth loss, coronal and root caries, and periodontal disease, regardless of other social and behavioral factors. CONCLUSIONS: Lifelong tobacco use is a modifiable risk factor for poor dental health among older adults. Dental practitioners need to intervene with all their adult patients to discourage use of tobacco products for oral as well as general preventive health care.

Relationship of Resting Lung Mechanics and Exercise Pattern of Breathing in Patients with Chronic Obstructive Lung Disease—JM Marin, SNA Hussain, WJ Gibbons, M Polverino, RD Levy, MG Cosio. Chest 1993;104:705.

To investigate the influence of resting pulmonary mechanics on the pattern of breathing during exercise in chronic obstructive pulmonary disease (COPD), we studied 29 patients with moderate to severe COPD (FEV_1 50 ± 20% predicted), and 10 normal subjects. Lung mechanics were studied using esophageal balloon technique and body-box. Incremental exercise testing was performed to exhaustion. As minute ventilation ($\dot{V}_E$) increases, COPD patients with the highest pulmonary resistance (R_L) or lowest elastic recoil pressure (P_L), used a greater tidal volume/vital capacity ratio (V_T/VC) than the COPD patients with more normal R_L or lowest P_L. To describe the breathing pattern during exercise, an exponential constant (K) describes the rates of increase in V_T/VC ratio with increasing $\dot{V}_E$, calculated according to the equation $V_T = VC(1 - e^{-K\dot{V}_E})$. The K values achieved by COPD patients were higher than in normal subjects. In addition, K value correlated negatively with the resting FEV_1 and FVC of COPD patients. When COPD patients were grouped according to their K values, it was revealed that patients with high K values generated greater V_T/VC ratio and also have the most abnormal resting lung mechanics. These results suggest that the exercise breathing pattern in COPD patients is significantly influenced by the degree of impairment of resting lung mechanics.

Long-Term Outcome in Patients Undergoing Surgical Repair of Tetralogy of Fallot—JG Murphy, BJ Gersh, DD Mair, V Fuster, MD McGoon, DM Ilstrup, et al. N Engl J Med 1993;329:593.

BACKGROUND: Although corrective surgery for Tetralogy of Fallot has been available for more than 30 years, the occurrence of late sudden death in patients in whom surgery was apparently successful remains worrisome. METHODS: We studied long-term survival among 163 patients who survived 30 days after complete repair of Tetralogy of Fallot, examining follow-up hospital records and death certificates when relevant. RESULTS: The overall 32-year actuarial survival rate among all patients who survived surgery was 86%, as compared with an expected rate of 96% in a control population matched for age and sex ($p < 0.01$). Thirty-year actuarial survival rates were calculated for the patient subgroups. The survival rates among patients less than 5 years old, 5 to 7 years old, and 8 to 11 years old were 90, 93, and 91%, respectively—slightly less than the expected rates ($p < 0.001$, $p = 0.06$, and $p = 0.02$). Among patients 12 years old or older at the time of surgery, the survival rate was 76%, as compared with an expected rate of 93% ($p < 0.001$). The performance of a palliative Blalock-Taussig shunt procedure before repair, unlike the performance of a Waterston or Potts shunt procedure, was not associated with reduced long-term survival, nor was the need for a transannular patch at the time of surgery. Independent predictors of long-term survival were older age at operation ($p = 0.02$) and a higher ratio of right ventricular to left ventricular systolic pressure after surgery ($p = 0.008$). Late sudden death from cardiac causes occurred in 10 patients during the 32-year period. CONCLUSIONS: Among patients with surgically repaired Tetralogy of Fallot, the rate of long-term survival after the postoperative period is excellent but remains lower than that in the general population. The risk of late sudden death is small.

Cigarette Smoking and Bone Mineral Density in Older Men and Women—KA Hollenbach, E Barrett-Connor, SL Edelstein, T Holbrook. Am J Public Health 1993;83:1265.

OBJECTIVES: The association between cigarette smoking and bone mineral density was examined prospectively in a population-based study of older Caucasian men and women. METHODS: Smoking patterns were determined at a 1972-1974 baseline evaluation and, again, 16 years later when 544 men and 822 women had bone mineral density measurements taken. RESULTS: Men and women who were cigarette smokers at baseline demonstrated significantly reduced bone mineral density of the hip compared with nonsmokers. Baseline smoking was not associated with significantly lower bone density at non-hip sites. Women demonstrated a significant dose-response relationship between baseline smoking status at all hip sites measured. Both sexes exhibited significant dose-response relationships between hip bone mineral density and change in smoking status between baseline and follow-up, demonstrating that smoking cessation in later life was ben-

eficial in halting bone density loss associated with smoking. CONCLUSIONS: Smoking was positively and significantly associated with decreased hip bone mineral density in old age. Bone loss associated with smoking would be expected to predict an increased risk of hip fracture in those who do not succumb earlier to another complication of tobacco use.

Project Towards No Tobacco Use: 1-Year Behavior Outcomes—S Sussman, CW Dent, AW Stacy, P Sun, S Craig, TR Simon, et al. Am J Public Health 1993;83:1245.

OBJECTIVES: We present 1-year follow-up data from a school-based tobacco use prevention project designed to test the effectiveness of three main components of social influence programs. The components teach refusal skills, awareness of social misperceptions about tobacco use, and misconceptions about physical consequences. METHODS: Four different curricula were developed and tested in a randomized experiment involving 48 junior high schools. The outcome variables examined were changes in initial and weekly cigarette and smokeless tobacco use 1 year after the intervention. RESULTS: Analyses indicated that each of the component programs were effective in decreasing both the initial and the weekly use of cigarettes except for the curriculum in which refusal skills were taught. Also, each curriculum was effective in decreasing the initial use of smokeless tobacco except for the one aimed at correcting social misperceptions. Only the combined curriculum showed an effect on the weekly use of smokeless tobacco. CONCLUSIONS: The combined intervention was the most effective overall in reducing the initial and weekly use of cigarettes and smokeless tobacco. This suggests that different reasons for use exist and need to be counteracted simultaneously. However, since single programs were also effective in reducing all but weekly smokeless tobacco use, any of these components may be worthwhile prevention tools.

Error in Smoking Measures: Effects of Intervention on Relations of Cotinine and Carbon Monoxide to Self-Reported Smoking—RP Murray, JE Connett, GG Lauger, and HT Voelker for the Lung Health Study Research Group. Am J Public Health 1993;83:1251.

OBJECTIVES: Sources of measurement error in assessing smoking status are examined. METHODS: The Lung Health Study, a randomized trial in 10 clinical centers, includes 3,923 participants in a smoking cessation program and 1,964 usual care participants. Smoking at first annual follow-up was assessed by salivary cotinine, expired air carbon monoxide, and self-report. Each of these measures is known to contain some error. Sensitivity and specificity were calculated by comparing a biochemical measure with self-report to produce an undifferentiated estimate of error. Classification error rates due to imprecision of the biochemical measures and to the error in self-report were estimated separately. RESULTS: For cotinine compared with self-report, the sensitivity was 99.0% and the specificity 91.5%. For carbon monoxide compared with self-report, the sensitivity was 93.7% and the specificity 87.2%. The classification error attributed to self-report, estimated by comparing the results from intervention and control groups, was associated with the responses of 3% and 5% of participants, indicating a small but significant bias toward a socially desirable response. CONCLUSIONS: In absolute terms in these data, both types of error were small.

The Long-Term Prevention of Tobacco Use Among Junior High School Students: Classroom and Telephone Interventions—JP Elder, M Wildey, C de Moor, JF Sallis Jr, L Eckhardt, C Edwards, et al. Am J Public Health 1993;83:1239.

OBJECTIVES: The purpose of this study was to implement and evaluate the effectiveness of a long-term tobacco use prevention program for junior high school students that used college undergraduate change agents and telephone boosters. METHODS: A psychosocial intervention combining refusal skills training, contingency management, and other tobacco use prevention methodologies such as telephone and mail boosters was implemented in 11 junior high schools in San Diego County, California. Eleven other junior high schools served as controls. Of the 2,668 participants, 57% were White/non-Hispanic, 24% were Hispanic, and 19% were of other racial/ethnic groups. College undergraduates served as change agents for both the classroom and booster interventions, the latter of which was delivered in the third (ninth-grade) year of the program. RESULTS: At the end of the third year, the prevalence of tobacco use within the past month was 14.2% among the intervention students and 22.5% among the controls, yielding an odds ratio of 0.71 for analysis at the school level. CONCLUSIONS: Both college undergraduate change agents and direct one-to-one telephone interventions appear to provide cost-effective tobacco-related behavior modification.

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New Horizons IX: Cost-Effectiveness of Respiratory Care in the ICU

In this issue of the Journal, we are pleased to feature two more papers[1] based on presentations from the Lifecare New Horizons Symposium presented at the AARC Annual Meeting in December 1993.

First is "Inhaled Bronchodilators during Mechanical Ventilation: Delivery Techniques, Evaluation of Response, and Cost-Effectiveness."[2] Dean Hess MEd RRT has masterfully collated much of the data that exist regarding alternatives and methods and costs of administering bronchodilators to intubated patients. The second of these papers, "Monitoring Gas Exchange: Clinical Effectiveness and Cost Considerations,"[3] by Charles G Durbin Jr MD, describes and discusses the application of various devices for monitoring the oxygenation and ventilation of patients in the intensive care unit. This paper explores the concepts of clinical efficacy, effectiveness, risk, and cost and focuses on overall usefulness of these monitors in the care of patients.

We present these papers because we believe that they are a valuable resource for clinicians who are responsible for providing clinically appropriate and cost-effective patient care. Bon appetit!

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Inhaled Bronchodilators during Mechanical Ventilation: Delivery Techniques, Evaluation of Response, and Cost-Effectiveness

Dean Hess MEd RRT

I. Introduction
II. Use of Small Volume Nebulizers during Mechanical Ventilation
III. Factors Affecting Deposition from Small Volume Nebulizers during Mechanical Ventilation
  A. Endotracheal Tube Size
  B. Nebulizer Placement
  C. Type of Nebulizer and Fill Volume
  D. Presence of Humidification Device
  E. Treatment Time
  F. Duty Cycle
  G. Mechanical Ventilator Brand
IV. Use of MDI during Mechanical Ventilation
V. MDI with Nozzle Extension
VI. Aerosol Delivery during Pediatric Mechanical Ventilation
VII. Assessment of Need
  A. History and Physical Examination
  B. Lung Mechanics
VIII. Assessment of Response
  A. Physical Findings
  B. Flow-Volume and Volume-Pressure Loops
  C. Airway Pressure and Lung Mechanics
  D. Resistance Using the Interrupter Technique
IX. Deposition and Dose
X. Cost
  A. MDI vs SVN
  B. Cost for Evaluation of Response
XI. In Summary

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A version of this paper was presented during the Lifecare New Horizons Symposium "Cost-Effectiveness of Respiratory Care in the ICU" at the 1993 Annual Meeting of the AARC in Nashville, Tennessee.

The author has no financial interest in any of the devices described.

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Introduction

Therapeutic aerosols are often used in mechanically ventilated patients, most commonly to administer β_2-agonist bronchodilators. Some aspects of this subject have been described in detail elsewhere.[1-3] In this paper I review techniques for aerosol administration during mechanical ventilation, discuss assessment of need and evaluation of response to β_2-agonist bronchodilator therapy in mechanically ventilated patients, and address the difficult subject of cost-effectiveness of these techniques.

Use of Small Volume Nebulizers during Mechanical Ventilation

MacIntyre et al[4] found that only 2.9% of a radioactive label placed into a small volume nebulizer (SVN) was deposited in the lungs of mechanically ventilated adult patients. Fuller et al[5] reported 1.22% deposition from an SVN in mechanically ventilated patients. However, the methodology of that study may have been flawed because of the radioactive tag used, and post-hoc adjustments to the data of Fuller et al suggest that deposition from the SVN in that study may have been as high as 3.66%.[6] Thomas et al[7] evaluated pulmonary aerosol deposition in 9 mechanically ventilated patients who had undergone open-heart surgery. They used the Servo 900C ventilator with a Siemens 945 nebulizer driver that powered the nebulizer only during inspiration to aerosolize 3 mL of radioactive solution (^{99m}Tc-human serum albumin) for 22 minutes. A mean of only 2.2% of the initial nebulizer dose was deposited in the lungs, and there was considerable intersubject variability. These data are in contrast to those from nonintubated patients in whom approximately 10% of the dose was deposited in the lungs.[8]

Although pulmonary deposition may be less in mechanically ventilated patients than in nonintubated patients, a physiologic response can be demonstrated in such patients. For example, Zandstra et al[9] found that nebulization of a mucolytic agent caused a significant increase in inspiratory airway resistance in a group of 10 postoperative intubated mechanically ventilated patients.

There are several disadvantages associated with the use of an SVN during mechanical ventilation. Contaminated medication nebulizers can be a source of bacteria-laden aerosols in mechanical ventilator circuits.[10,11] The continuous flow from an SVN increases the tidal volume delivered (and associated pressure) when volume-targeted ventilation is used.[12] The continuous flow from an SVN introduces a bias flow that makes it more difficult for the patient to generate the trigger pressure required during patient-triggered modes of ventilation (eg, pressure support, assist/control).[13] The continuous flow of aerosol from the SVN can also damage the expiratory flow transducer of some pneumotachometer-equipped ventilators. It is a common practice to place filters in the expiratory limb to protect the flow transducer during aerosol administration; however, obstruction of these filters can retard exhalation and result in air trapping.

Factors Affecting Deposition from SVNs during Mechanical Ventilation

Endotracheal Tube Size

Using a bench model, Ahrens et al[14] showed that the endotracheal tube is a formidable barrier to the penetration of aerosols from an SVN into the lower respiratory tract. They found that the amount of aerosol deposition in a lung model decreased as the endotracheal tube size decreased and as inspiratory flow increased. However, O'Riordan et al[6] found no difference in deposition between models with 7.0- and 9.0-mm adult endotracheal tubes.

Nebulizer Placement

When an SVN is used in a ventilator circuit, it can be placed at the ventilator Y-piece or at some point in the inspiratory circuit between the ventilator and the Y-piece. Also, the nebulizer can be powered continuously from an auxiliary gas flow or intermittently from the nebulizer drive line of the ventilator. Using a bench model, Hughes and Saez[15] found that significantly more medication was delivered to the endotracheal tube if the nebulizer was placed at the manifold (approximately 18 inches from the Y-piece) and operated during inspiration only. The least amount of aerosol was delivered to the endotracheal tube when the nebulizer was placed at the Y-piece and operated continuously—a

technique commonly used in clinical practice. Quinn[16] also found that placing the SVN closer to the ventilator (as opposed to placing it at the Y-piece) improved the efficiency—% total dose deposited—of aerosol delivery. Unlike Hughes and Saez, however, Quinn did not find that intermittent nebulization (ie, inspiration only) improved efficiency. Continuous nebulization (ie, throughout the respiratory cycle) and placement of the nebulizer at the manifold of the ventilator circuit presumably allows the inspiratory tubing to fill with aerosol during the expiratory phase of the ventilator. A system for medication delivery during mechanical ventilation that delivers aerosol into the inspiratory circuit only during the expiratory phase has been described.[17]

Type of Nebulizer and Fill Volume

Nebulizer brand and fill volume affect SVN performance[18-24] whether the patient is breathing spontaneously or is being mechanically ventilated. It has been suggested that the choice of an SVN brand may be the most important determinant of aerosol deposition in the lower respiratory tract.[6]

Presence of a Humidification Device

The inclusion of a humidifier in the ventilator circuit decreases aerosol delivery by 40-50%.[6] Therefore, it is probably desirable to bypass the humidifier during therapeutic aerosol administration. If an artificial nose is used for humidification, it must be removed from the ventilator circuit during aerosol therapy.

Treatment Time

It is reasonable to expect greater pulmonary aerosol delivery with longer treatment times.[5] Continuous nebulization can be used during mechanical ventilation in patients with severe acute reversible airflow obstruction.[25-29] Virtually all published reports of continuous nebulization have been of spontaneously breathing nonintubated patients, and most of these data are from pediatric patients with asthma. A controlled study of continuous versus intermittent nebulization in spontaneously breathing children with asthma has shown that continuous nebulization is superior to intermittent nebulizer treatment.[29] In this study, dose was controlled (equal hourly dose in both treatment groups), suggesting that delivery technique (continuous vs intermittent) may be an important determinant of patient response. Controlled studies are needed to evaluate the effect of continuous nebulization in mechanically ventilated patients with acute bronchospasm. When continuously aerosolized β_2 agonists are administered, it is important to carefully monitor the patient for both beneficial and adverse effects. In particular, the patient should be monitored for the presence of arrhythmias, hypokalemia, and tremor.[30]

Duty Cycle

O'Riordan et al found that increasing the duty cycle (ie, the ratio of inspiratory time to total cycle time or t_I/t_{tot}) significantly increased aerosol delivery to a lung model.[6] Similarly, O'Doherty et al[31] found that aerosol delivery was augmented by increasing the inspiratory time and decreasing the respiratory rate.

Mechanical Ventilator Brand

Some commercially available mechanical ventilator systems have integral nebulizer drive systems. When these systems are used, the ventilator pressurizes the nebulizer during the inspiratory phase. Advantages of using the ventilator to power the nebulizer include elimination of the need for an external gas source, stable F_{IO_2} and tidal volume delivery during the nebulizer treatment, and maintenance of sensitivity (ie, ability of the patient to trigger the ventilator). An important disadvantage of using this method is increased time required for nebulization. Current ventilator systems pressurize the nebulizer drive line to 5-10 psi, and provide a duration of nebulization of 15-30 min. McPeck et al[32] evaluated the performance of 4 ventilator brands on nebulizer performance, and found that 3 of the 4 ventilator systems delivered approximately 10-15% of the initial nebulizer dose in a 30-min period (Fig. 1). However, one of the ventilator systems (the Veolar, Hamilton Medical, Reno NV) delivered < 5% of the dose in a 90-min nebulization time.

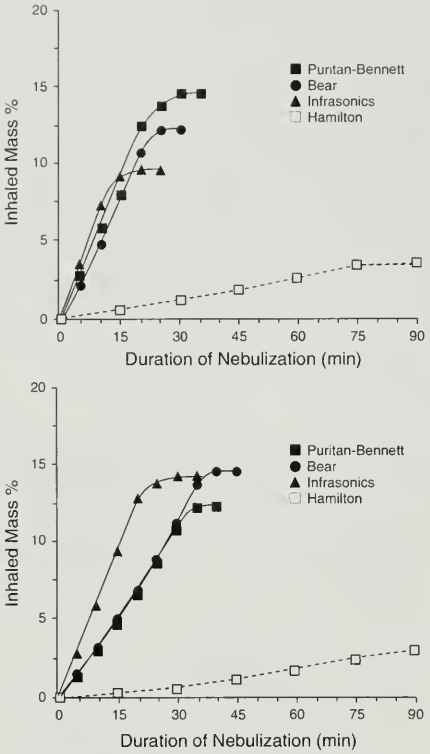

Fig. 1. Inhaled mass% (% of initial nebulizer charge) over time measured at the distal end of the ETT (upper panel = Airlife nebulizer; lower panel = Hudson nebulizer). Measurements of radioactivity were made at 5-minute intervals for all ventilators except the Hamilton, which was measured at 15-minute intervals. Nebulization to dryness is indicated by the development of a plateau. (From Reference 32.)

Use of MDI during Mechanical Ventilation

Several bench studies have evaluated the delivery of aerosols by metered-dose inhalers (MDI) through endotracheal tubes. Crogan and Bishop,[33] using an MDI adapter attached between the ventilator Y-piece and a swivel adapter attached to the endotracheal tube, found that significantly more aerosol traversed the endotracheal tube when the MDI was activated into a continuous flow of gas (analogous to actuation of the MDI near the beginning of the inspiratory phase of the ventilator). They also found that the amount of aerosol that passed through the endotracheal tube varied with endotracheal tube size: 3.0% for a 6.0-mm tube and 6.5% for a 9.0-mm tube. My co-workers and I[34] found that 3.3% of the aerosol from an MDI traversed the endotracheal tube, 7.4% impacted on the MDI adapter, and 89.3% impacted on the endotracheal tube. Flow, tidal volume, and endotracheal tube size did not affect the amount of aerosol that passed through the endotracheal tube; however, only two tube sizes were used (7.0 mm and 9.0 mm). Using a lung model, Fuller et al[35] found significantly more aerosol delivery from an MDI with a holding chamber than from a nebulizer.

Several studies have evaluated the use of an MDI in mechanically ventilated adult patients. Gay et al[36] compared the response to albuterol by MDI and SVN. The nebulizer was placed at the Y-piece of the ventilator, and each puff from the MDI was administered during a slow inspiration with the lungs held at hyperinflation for several seconds. All 18 of the patients had an increase in expiratory flow in response to at least one of the administration methods; three patients responded only to SVN, and one responded only to MDI. Fernandez et al[37] compared the use of salbutamol by MDI, ipratropium bromide by MDI, and intravenous aminophylline in mechanically ventilated patients with COPD. They found that these three drugs were equally effective in producing bronchodilatation and concluded that ipratropium bromide and salbutamol administered by MDI were as effective as aminophylline administered intravenously. Fuller et al[5] compared the delivery of labeled fenoterol via MDI and SVN in mechanically ventilated patients, and found a significantly greater deposition with MDI (5.76%) than with SVN (1.22%). However, SVN deposition may have been as high as 3.66% if the data are adjusted for the methodologic effect of the radiolabel used.[6]

There are several commercially available adapters that allow an MDI to be placed into a ventilator circuit.[1,38] These include chamber (reservoir, spacer) devices, in-line devices, and elbow devices. The chamber-spacer devices fit into the inspiratory limb of the circuit and act as an aerosol reservoir. This action should allow the high initial velocity of aerosol particles to slow and the particle size to decrease. The in-line devices can be attached at any point along the inspiratory limb of the circuit, and

can be positioned in the inspiratory limb in such a way as to use the ventilator circuit as a reservoir. The elbow devices attach directly to the endotracheal tube and direct the aerosol into the endotracheal tube during inspiration.

Ebert et al[38] compared 9 MDI adapters, and found that the chamber-style devices produced greater respirable volumes of aerosol than the in-line or elbow devices (Fig. 2). Rau et al[39] compared a reservoir chamber device to an elbow device, and found that significantly more aerosol was delivered through the endotracheal tube when the chamber device was used. Bishop et al[40] compared the volume of particles delivered to the distal end of the endotracheal tube with several different MDI adapters. They found that each of the ventilator adapters delivered less aerosol through the endotracheal tube than from the actuator supplied by the manufacturer for use with ambulatory patients. They also found that the greatest amount of aerosol was delivered through the endotracheal tube using a reservoir device, the least amount was delivered using an elbow device, and an intermediate amount was delivered using an in-line device. Because these were bench studies, the clinical implications are not completely clear, but it could mean that the dose (number of puffs) may need to be adjusted based upon the MDI adapter that is used. Clinical studies are needed to confirm this speculation.

There are sufficient data to conclude that a measurable amount of aerosol is delivered through the endotracheal tube when an MDI is used, and that the

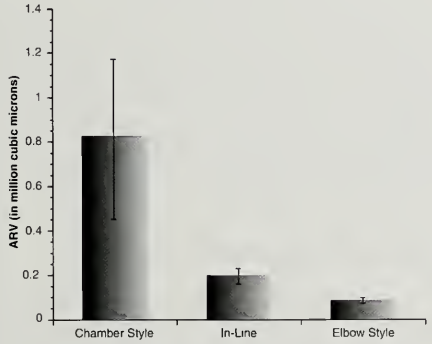

Fig. 2. Mean available respirable volumes (with standard deviation bars) measured after use of the three styles of MDI spacers and adapters tested. (From Reference 38.)

physiologic response of an intubated patient is similar whether an MDI or an SVN is used. Use of MDI during mechanical ventilation avoids the problems associated with SVNs. Use of MDI rather than SVN during mechanical ventilation may also decrease the cost of therapy and require less time.[1,41,42]

The MDI is an effective substitute for an SVN in intubated, mechanically ventilated adult patients. It can be administered using a closed system (ie, the MDI adapter is a permanent part of the ventilator circuit), and the MDI adds very little volume to the system when it is actuated.[43] However, it must be recognized that the optimal ventilator settings for this therapy are still unknown and much more clinical investigation is needed on this subject.

MDI with Nozzle Extension

One strategy used to improve the delivery of aerosolized drug from an MDI is to extend the nozzle of the MDI canister to the distal tip of the endotracheal tube. A simple and convenient way of doing this is to attach a 19-gauge vascular catheter to a 60-mL syringe (Fig. 3).[44] The catheter can then be passed through the endotracheal tube, and the MDI can be actuated by pressing the syringe plunger.

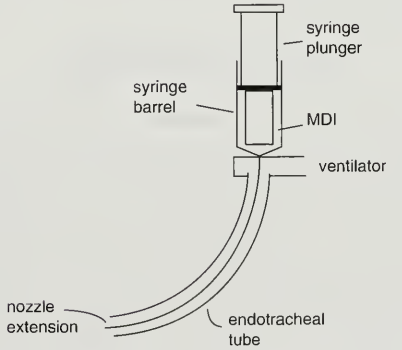

Fig. 3. Schema of system to deliver aerosolized medication from an MDI using a nozzle extension.

Taylor and Lerman[45] evaluated the delivery of MDI albuterol through pediatric endotracheal tubes using an elbow adapter and a 19-gauge catheter as a nozzle extension passed to the distal end of the endotracheal tube. Using this configuration, 96.7% of

the dose was delivered to a filter placed at the distal end of the endotracheal tube. Similar results were reported by Niven et al,[46] who found that small bore extensions (< 1-mm internal diameter), which traverse the length of a standard adult endotracheal tube, can deliver about 90% of the nominal MDI dose beyond the endotracheal tube tip. In that study, there was a reduction in distal delivery when the inner diameter of the catheter exceeded 1 mm. In contrast to these results, Taylor et al[47] found little difference in delivered dose with 14-, 19-, and 22-gauge catheters. They also found that the effect of the nozzle extension was lost if it did not pass to the distal end of the endotracheal tube (Fig. 3).

Although bench studies using MDI nozzle extensions through endotracheal tubes are promising, no clinical evaluations of this method have yet been published. Accordingly, this method should be considered experimental, and it should not be used without appropriate institution-approved research protocols. Several studies[48,49] in a rabbit model suggest that albuterol delivery using this method may induce serious tracheal epithelial injury. However, 20 actuations from the MDI were used in that study. Because nozzle extensions result in a greater fraction of aerosol delivered through the endotracheal tubes, doses much lower than this are likely to be effective.

Aerosol Delivery during Pediatric Mechanical Ventilation

Brudno et al[50] administered aerosolized terbutaline to eight ventilator-dependent infants with bronchopulmonary dysplasia (BPD), and found a significant reduction in inspiratory and expiratory resistance and a significant increase in tidal volume as a result. Wilkie and Bryan[51] administered aerosolized salbutamol and ipratropium bromide to 17 ventilator-dependent infants with BPD (age range 19-103 days), and found a significant reduction in resistance and a significant improvement in pulmonary compliance in response to treatment. Although intermittent nebulization during mechanical ventilation has been shown to be superior to continuous nebulization during manual ventilation for pediatric patients,[52] the aerosol treatments were administered during manual ventilation in both of these studies.

Cameron et al[53] evaluated nebulizer function in neonatal ventilator circuits using a lung model consisting of a 3.5-mm endotracheal tube, a filter at the distal end of the endotracheal tube, and a time-cycled pressure-limited ventilator. A filter deposition (simulating lung deposition) of 1.52% at best was reported. The authors attributed this poor performance to the continuous flow through the ventilator circuit and presumed that much of the aerosol is carried out through the expiratory port of the ventilator by this flow. Flavin et al[54] used a rabbit model with an infant ventilator to simulate aerosol delivery to intubated mechanically ventilated infants and found that the amount of aerosol deposited in the lungs was extremely low (1.96% at best). Using a bench model with a 3.0-mm endotracheal tube and a pediatric ventilator, Rau and Harwood[55] reported a deposition at the end of the endotracheal tube of 1.78% with a 50-mL reservoir between the nebulizer and the Y-piece on the inspiratory limb of the circuit; the deposition was 0.97% when the nebulizer was placed just proximal to the Y-piece.

Although an MDI can be used effectively by many nonintubated children, few data exist regarding the use of MDI in intubated mechanically ventilated children. Recent studies[56,57] suggest that MDIs might be used effectively in mechanically ventilated infants. However, because of the limited data available regarding the use of MDIs during pediatric mechanical ventilation, they should be used cautiously in this patient population.

Assessment of Need

In many critical care units, it is assumed that all mechanically ventilated patients need inhaled bronchodilator therapy. Many ventilated patients receive inhaled bronchodilator, even though they likely would not otherwise receive such therapy. Ideally, inhaled bronchodilator should be reserved for those who are likely to benefit from this therapy.

History and Physical Examination

It is reasonable to administer inhaled bronchodilators to mechanically ventilated patients with a history of obstructive lung disease, particularly if such agents were part of the patient's treatment prior to intubation. The use of inhaled bronchodila-

tors in other acute disease states is less clear. Although it has been shown that airway resistance is increased in patients with the adult respiratory distress syndrome (ARDS),[58-62] it is unclear whether that increased resistance is responsive to bronchodilator administration. The increased airway resistance observed in patients with ARDS might be the result of decreased lung volume rather than a reversible reduction in airway caliber, which would make bronchodilator responsiveness unlikely.[62] Interestingly, Gay et al[63] found that 10 of 13 mechanically ventilated patients showed a decrease in airway resistance in response to inhaled metaproterenol, although only 3 were asthmatics who had previously used bronchodilators.

Auscultation may be useful to determine the need for inhaled bronchodilator therapy. Wheezing suggests the presence of bronchospasm, and a trial of inhaled bronchodilators is warranted. However, it must be remembered that "not all that wheezes is asthma." Wheezing secondary to pulmonary edema is not likely to respond to inhaled bronchodilators.

Lung Mechanics

A variety of both direct and calculated lung mechanics measurements can be used to assess bronchodilator response in mechanically ventilated patients. Unfortunately, baseline lung mechanics are often not useful to determine the need for inhaled bronchodilators. Unlike spirometric values, normal values for mechanical function, such as airway resistance, are not known for intubated patients. Further, resistance, compliance, and proximal airway pressure are affected not only by lung function but also by endotracheal tube size and ventilator settings. In spite of these limitations of lung mechanics measurements in ventilated patients, if a trial of inhaled bronchodilator demonstrates an improvement in lung mechanics, then such therapy should be continued.

Assessment of Response

Physical Findings

Just as auscultation may be useful to determine the need for inhaled bronchodilator, it can be used to assess response to therapy. An improvement in airflow and a decrease in wheezing are indicative of a positive bronchodilator response.

Flow-Volume and Volume-Pressure Loops

Passive flow-volume and volume-pressure loops may be used to evaluate bronchodilator response in some patients. An increase in expiratory flow and a decrease in resistive work indicates a positive bronchodilator response (Fig. 4). Several commercially available systems are capable of evaluating lung mechanics in mechanically ventilated patients. Similar systems are also available integral to current generation microprocessor ventilators. The slope of the expiratory flow-volume curve can be used to determine airway resistance. Using anesthetized cats, Zin et al[64] showed that the slope of the flow-volume

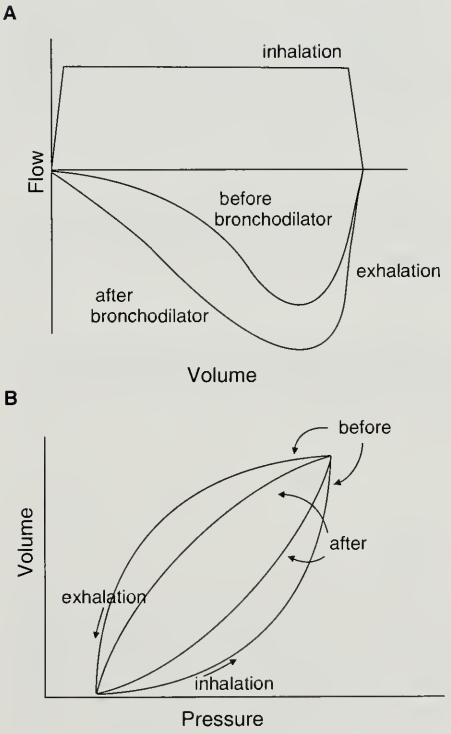

Fig. 4. Effect of inhaled bronchodilator on inspiratory flow-volume curve (A) and volume-pressure loop (B).

curve represents the time constant of the respiratory system (τ). Resistance (R) can then be calculated as

$$R = \tau/\text{compliance}.$$

Airway Pressure and Lung Mechanics

According to the equation of motion, the pressure applied across the respiratory system at any time during the ventilatory cycle—transpulmonary pressure (P_T)—is the sum of elastic pressure (P_{el}), resistive pressure (P_R), and positive end-expiratory pressure (PEEP).

$$P_T = P_{el} + P_R + PEEP.$$

Transpulmonary pressure is the sum of pressure provided by the ventilator and that provided by the respiratory muscles. It can be estimated from the proximal airway pressure during passive mechanical ventilation, but requires the use of an esophageal balloon during active inspiration and spontaneous breathing modes. At end-inspiration, P_{el} is determined by tidal volume (V_T) and compliance (C):

$$P_{el} = \frac{V_T}{C}.$$

P_R at end-inspiration is determined by flow ($\dot{V}$) and inspiratory resistance (R_I):

$$P_R = \dot{V} \cdot R_I.$$

The total PEEP level ($PEEP_{tot}$) is the sum of the set-PEEP and auto-PEEP. Auto-PEEP is determined by V_T, C, expiratory resistance (R_E), and expiratory time (t_E).[65] That is,

$$\text{auto-PEEP} = \frac{V_T}{C\,(e^{K_E t_E} - 1)},$$

$$\text{where } K_E = \frac{1}{R_E \cdot C}.$$

During constant-flow, volume-targeted ventilation, P_{el}, P_R, and $PEEP_{tot}$ can be determined by applying an end-inspiratory and an end-expiratory pause. An end-inspiratory pause of sufficient duration (0.5-2.0 s) will allow equilibration of proximal airway pressure and peak alveolar pressure, and is commonly called plateau pressure (P_{plat}). This can be achieved on most ventilators by use of the end-inspiratory pause control. This measurement should be made on a single breath and the pause removed immediately afterward. Otherwise, plateau pressure and peak inspiratory pressure increase due to the prolonged inspiratory time, and auto-PEEP may result. An end-expiratory pause can be applied on some ventilators by use of the expiratory-hold control. For ventilators that do not allow this, auto-PEEP can be measured by use of a Braschi valve (Fig. 5). Although inspiratory flow has little effect on P_{el}, there is a curvilinear increase in P_R with an increase in flow.[66]

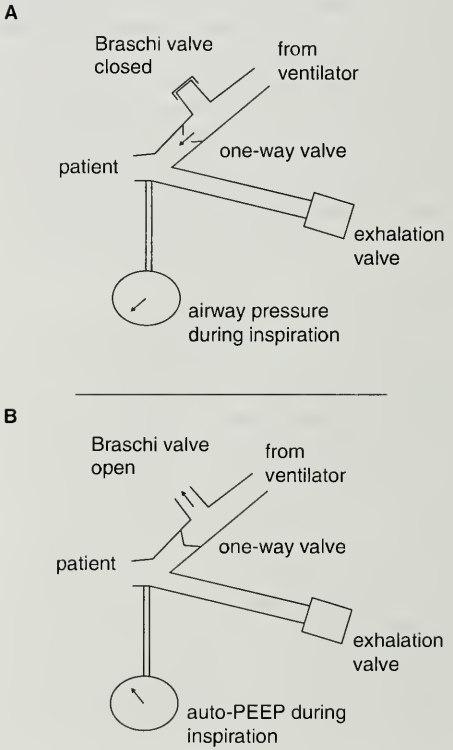

Fig. 5. Use of Braschi valve to measure auto-PEEP. With Braschi valve closed (A), the ventilator works normally. With Braschi valve open (B), an end-expiratory pause equal in duration to the inspiratory time is created.

The relationships among peak inspiratory pressure (P_{max}), P_{plat}, PEEP, and $PEEP_{tot}$ are illustrated in Figure 6. Note that

$$P_R = P_{max} - P_{plat}, \text{ and } P_{el} = P_{plat} - PEEP_{tot}.$$

Thus, R_I can be calculated by

$$R_I = \frac{P_{max} - P_{plat}}{\dot{V}_I},$$

and C can be calculated by

$$C = \frac{V_T}{P_{plat} - PEEP_{tot}}.$$

These relationships assume that resistance and compliance are linear over the range of tidal volume—an assumption that is not always valid. Also, these are global indices of resistance and compliance, and do not account for the inequality of regional time constants throughout the lungs.

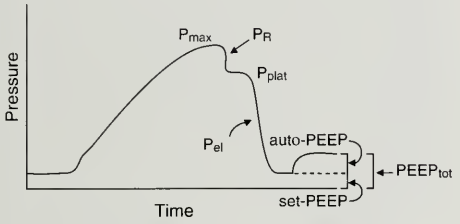

Fig. 6. Airway pressure waveform illustrating plateau pressure (end-inspiratory pause) and auto-PEEP (end-expiratory pause).

An important determinant of auto-PEEP is R_E, and administration of an inhaled bronchodilator should decrease R_E. Thus, an improvement in R_E following inhaled bronchodilator administration is likely to decrease auto-PEEP. This may be of limited usefulness, however, because auto-PEEP may not be present even if R_E is high, provided that t_E is long enough to allow complete exhalation. If auto-PEEP decreases following bronchodilator administration, compliance may increase.

Gay et al[63] evaluated inhaled bronchodilator responsiveness in 13 mechanically ventilated patients by administration of 1.8 mg of metaproterenol. Following bronchodilator administration, they found decreases in P_{max}, P_{el}, P_R, and auto-PEEP (Fig. 7). They also found that the decrease in auto-PEEP was associated with a decrease in end-expiratory lung volume of 0.2 L. The decrease in P_{max} was likely due to decreases in P_{el}, P_R, and auto-PEEP. They concluded that P_{max} and auto-PEEP are useful indicators of bronchodilator-induced changes in lung function.

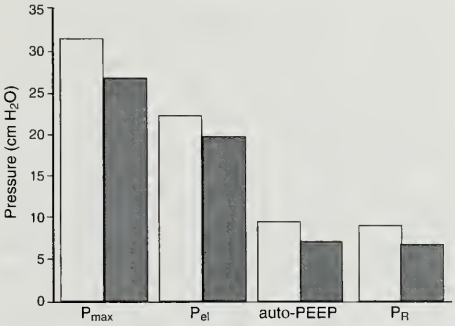

Fig. 7. Airway pressure measurements before (□) and after (■) administration of inhaled bronchodilator (based on data from Reference 63).

My co-workers and I[67] evaluated the usefulness of a variety of lung mechanics measurements on response to four puffs of albuterol in 21 mechanically ventilated patients. A bronchodilator response could be demonstrated in 14 of the 21 patients, and 6 of the 14 responders had a history of COPD (7 were receiving other bronchodilator therapy). The largest changes following bronchodilator administration were observed for P_{max} and R_E (Table 1). P_R was insensitive to bronchodilator response, and auto-PEEP was not useful because it was zero in all patients.

Table 1. Changes in Lung Mechanics with Inhaled Albuterol (4 Puffs) in 14 Mechanically Ventilated Patients[67]

	Before Albuterol	After Albuterol	% Change
P_{max} (cm H_2O)	33	29	12.1
P_R (cm H_2O)	9	8	11.1
P_{el} (cm H_2O)	26	23	11.5
R_E (cm $H_2O \cdot L^{-1} \cdot s$)	28.7	23.1	19.5

During pressure-targeted ventilation, changes in P_{max} and P_R are not useful to evaluate changes in airway resistance. P_{max} is preset and should not change. P_R is zero because pressure-targeted ventilation results in near-zero end-inspiratory $\dot{V}_I$ (note that $P_R = \dot{V} \cdot R_I = 0 \cdot R_I = 0$). With this mode of ventilation, V_T is dependent upon R_I and R_E,[65] as shown by

$$V_T = \frac{P_{set}(e^{K_E t_E} - 1)(e^{K_I t_I} - 1)}{e^{K_E t_E + K_I t_I} - 1},$$

where $K_E = \frac{1}{R_E \cdot C}$ and $K_I = \frac{1}{R_I \cdot C}$.

For example, assume that P_{set} = 25 cm H_2O, t_I = 1 s, t_E = 2 s, C = 0.03 L/cm H_2O, R_I = 20 cm $H_2O \cdot L^{-1} \cdot s$, and R_E = 30 cm $H_2O \cdot L^{-1} \cdot s$. This should produce V_T = 0.55 L. If an inhaled bronchodilator decreases R_I to 15 cm $H_2O \cdot L^{-1} \cdot s$ and R_E to 20 cm $H_2O \cdot L^{-1} \cdot s$, then V_T should increase to 0.65 L.

The resistance to flow through endotracheal tubes can be a significant contributor to P_{max} and P_R (Table 1).[66,68-78] This might complicate the evaluation of bronchodilator response because small changes in airway resistance may be masked by the relatively large fixed resistance through the endotracheal tube. Techniques have been described to mathematically correct for endotracheal tube resistance (Table 2),[68] but these are not easily applied without the use of a computer.

Table 2. Pressure Drop across an 8-mm Endotracheal Tube at 1 L/s Flow[68]*

First Author	Pressure Drop (cm H_2O)
Sahn[69]	1.9
Sullivan[70]	6.1
Behrakis[71]	9.8
Gottfried[72]	5.9
Rossi[66]	5.9
Weissman[73]	4
Wright[74]	4.1
Wright[74] (patients)	5.3
Prezant[75]	5.5
van den Berg[76]	11.1†
Pesenti[77] (patients)	8.9
Bhatt[78]	5.4

*Unless otherwise indicated, data are from a physical laboratory model.
†Includes ventilator tubing.

Techniques are also available to calculate expiratory resistance.[79] One method is that described by Jonson et al,[80] in which P_{el} is divided by peak expiratory flow ($\dot{V}_{E\,max}$):

$$R_E = \frac{P_{plat} - PEEP_{tot}}{\dot{V}_{E\,max}}.$$

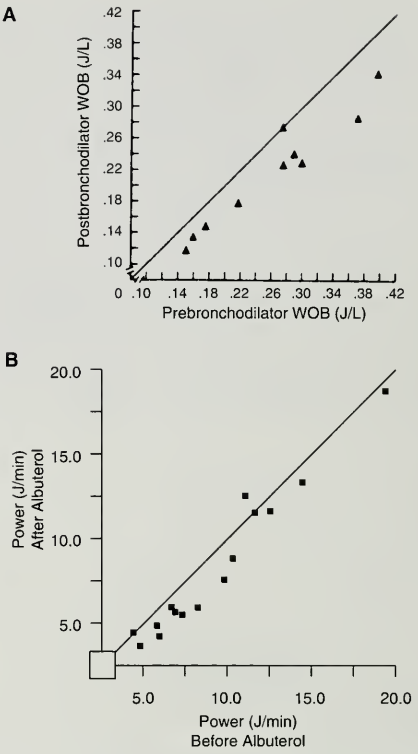

Fig. 8. Change in work of breathing following inhaled bronchodilator in intubated patients. (A) Inspiratory work performed by the ventilator before and after administration of bronchodilator. All but one patient showed a decrease in work after bronchodilator (adapted from Reference 63, with permission). (B) Plot of the individual values of the total work of breathing, expressed as power (J/min), before inhalation of albuterol on the horizontal axis against values obtained after albuterol inhalation on the vertical axis (adapted from Reference 82, with permission). The solid lines represent the line of identity.

R_E is typically greater than R_I[79,81] and may be more useful than R_I to evaluate bronchodilator response. However, this has yet to be evaluated and reported in the peer-reviewed literature.

Inspiratory work of breathing (WOB) can be determined by integration of the area under the volume-pressure curve. An increase in airway resistance should increase WOB, and WOB might be expected to decrease with a response to inhaled bronchodilator. During passive mechanical ventila-

tion, WOB can be estimated from measurements of pressure and flow at the proximal airway. Gay et al[63] found a decrease in WOB following inhaled bronchodilator administration (Fig. 8), as did Mancebo et al[82] and my group.[67]

Resistance Using the Interrupter Technique

The interrupter technique uses measurements of pressure and flow at the airway to estimate respiratory mechanics.[60,61,66,72,83-86] During expiration, flow is suddenly interrupted. Pressure is measured after interruption, and flow is measured immediately prior to the interruption. The interruptions are repeated rapidly, allowing multiple measurements during a single breath. The compliance of the respiratory system is determined by plotting postinterruption pressure against the corresponding volume above the end-expiratory level (Fig. 9). The slope of this relationship represents compliance. The resistance of the respiratory system can be determined by plotting the plateau in postinterruption pressure against the flow immediately prior to the interruption (Fig. 10). The slope of this relationship represents resistance.

An interrupter technique was used by Gay et al[36] to compare MDI and SVN in mechanically ventilated patients. Recordings were made during stepwise deflations of the relaxed respiratory system (Fig. 11). The recoil pressures and corresponding expiratory flows were plotted (Fig. 12). Because this relationship is nonlinear in patients with dynamic airway collapse, flows at recoil pressures of 6 and 10 cm H_2O (iso-recoil flow) were averaged ($\dot{V}_{Pmean}$) from linear approximations of the data. They found that albuterol resulted in an increased iso-recoil flow in all patients (Fig. 13).

Deposition and Dose

An interesting paradox exists when the dosages used with SVN and MDI are compared. The usual dose used with an SVN is approximately ten times the dose used with an MDI. For example, a 0.5-mL dose of albuterol (0.5 % solution) is equivalent to 2.5 mg, whereas two puffs by MDI (90 μg each) are equivalent to 0.18 mg. The dose from an SVN may need to be greater to compensate for the volume of drug retained in the dead volume of the nebulizer and the amount of drug lost during the expiratory phase (because most nebulizers are operated with

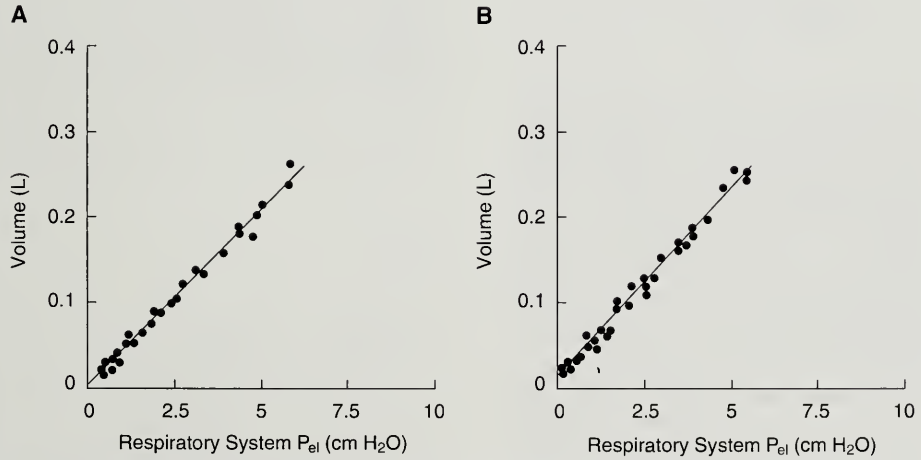

Fig. 9. Volume-pressure relationship determined by interrupter technique; slope represents compliance. (A) Data are from patients receiving anesthesia only. (B) Data are from patients receiving anesthesia and paralysis. Note that compliance is increased during anesthesia with paralysis as shown by the line shifted left. (Adapted from Reference 83, with permission.)

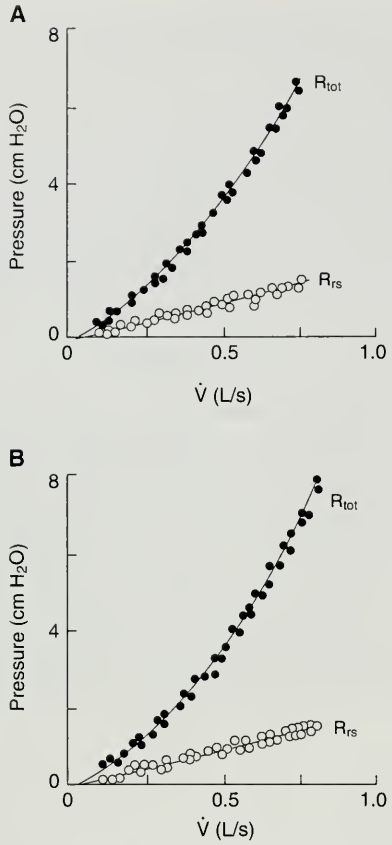

Fig. 10. Pressure-flow relationship (resistance) determined by interrupter technique. R_{tot} includes endotracheal tube and equipment, R_{rs} is of respiratory system only. A = anesthesia only; B = anesthesia + paralysis. (Adapted from Reference 83, with permission.)

continuous flow). In nonintubated patients, however, it has been shown that pulmonary deposition from the SVN and MDI is roughly equivalent (about 10% for each).[8,87]

Because the usual dose from the MDI is only a fraction of that with an SVN and the deposition from each is similar, more drug may be deposited in the lungs with the SVN. Using histamine bronchoprovocation as a bioassay, Blake et al[88] found that the standard dose of nebulizer solution (2.5 mg) delivered more albuterol to β_2 receptors than the standard two puffs of an MDI (0.18 mg). Moreover, data

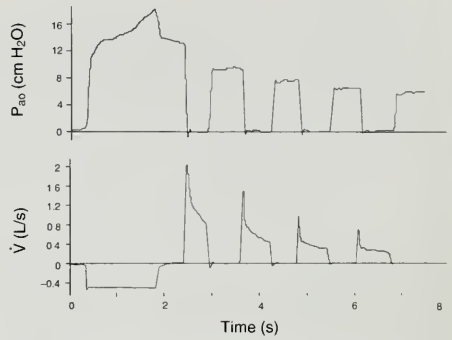

Fig. 11. Multiple flow interruptions during a single exhalation. P_{ao} = pressure measured at the airway opening. (Adapted from Reference 36, with permission.)

from this study suggest that 10 puffs from an MDI would be required to deliver the same amount of albuterol to β_2 receptors. This is consistent with several clinical studies that have reported greater effectiveness of inhaled β_2 agonist from SVN than MDI in acutely ill asthmatics.[89,90]

An unresolved issue relates to the dose when MDIs are used with intubated patients. Fernandez et al[37] used two puffs, Gay et al[36] used three puffs, and Fuller et al used four puffs.[5] Because the MDI dose deposited in the lungs is approximately half of the dose deposited in the lungs of nonintubated patients (Fig. 14), it seems reasonable to at least double the number of MDI puffs in intubated patients (ie, at least four puffs). In some patients, this dose will be inadequate, and a greater number of puffs (eg, 10-

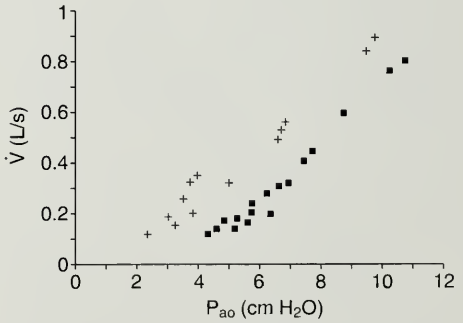

Fig. 12. Recoil flow-pressure relationships before (■) and after (+) inhaled bronchodilators. P_{ao} = pressure measured at the airway opening. (Adapted from Reference 36, with permission.)

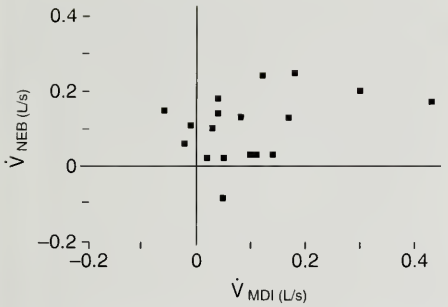

Fig. 13. Changes in iso-recoil flow with nebulizer and inhaler. Note that most patients show improvement with either technique. (From Reference 36, with permission.)

20) can be safely and effectively used.[3] These dose increases should be titrated to carefully monitored objective responses. With high-dose β_2-agonist therapy, the patient must be properly and continuously monitored for side effects such as cardiac arrhythmias, tremor, and hypokalemia. Such patients should also be treated comprehensively with systemic and other therapies to correct airway dysfunction (eg, steroids). In stable asthmatics, Maesen et al[91] studied the effects of maximal doses of β_2 agonist from an MDI on pulse rates, ECG, and serum potassium concentrations. For salbutamol, they found a maximal individual dose of 1,400 μg (14 puffs) in 3 patients, 2,200 μg in 8 patients, 3,000 μg in 1 patient, and 3,800 μg in 1 patient. At the maximal dose, they found no clinically important ECG changes, a small increase in pulse rate, and a small decrease in serum potassium concentrations. They concluded that high doses of β_2 agonists from an MDI were safe for patients.

Cost

MDI vs SVN

Cost comparisons of MDI vs SVN therapy have been reported. In nonintubated patients, there is clearly a cost savings with the use of MDI compared to SVN.[41,42] The savings are largely due to less personnel time required for MDI administration. Following initial instruction, patients can frequently self-administer MDI without the need for a therapist or nurse to supervise each treatment session. In one report, the hospital cost was $165.50/patient/week for SVN and $43.36/patient/week for MDI.[42]

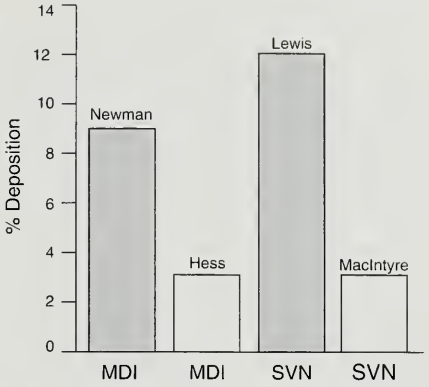

Fig. 14. Deposition with MDI and SVN, with (□) and without (■) endotracheal tube. (From data in References 4, 8, 34, and 87.)

Cost comparisons for MDI vs SVN have also been reported for mechanically ventilated patients.[1,5,42] The cost of an MDI treatment for mechanically ventilated patients has been estimated at about $2.50, and that for an SVN treatment at about $4.80. Much of this difference is related to therapist time. If one considers only drugs and materials, the cost/treatment is similar for MDI and SVN (both less than $0.50). For mechanically ventilated patients, it might be argued that therapist time to administer the treatment is of little importance because for either MDI or SVN the therapist must be in attendance (the mechanically ventilated patient is not likely to self-administer the MDI). Further, the therapist can be attending to other aspects of the patient-ventilator interface at the time of the treatment. When a large number of actuations from the MDI is required to achieve the desired effect, use of an MDI might actually be more expensive than an SVN.

Assessment of the cost of a procedure is difficult and institution specific. Costs of materials and personnel vary considerably from one hospital to another. When conducting a cost analysis, it is very important to consider actual costs to the hospital and not charges.[92] Patient charges are not a good proxy for costs, and are particularly meaningless with the widespread use of prospective payment.

Cost for Evaluation of Response

As have been described previously in this paper, there are many techniques that can be used to evaluate bronchodilator response in mechanically ventilated patients. Some methods are low tech-low cost and others are high tech-high cost (Table 3). The more sophisticated approaches may be more sensitive to detection of a bronchodilator response but are also, unfortunately, more expensive.

Table 3. Techniques To Evaluate Bronchodilator Response in Mechanically Ventilated Patients

Low Tech-Low Cost	High Tech-High Cost
Patient history	Airway resistance
Auscultation	Work of breathing
P_{max}	Expiratory flow-volume curves
$P_{max} - P_{plat} = P_R$	
V_T (pressure-targeted ventilation)	
Auto-PEEP	

It would seem appropriate and proper to evaluate the mechanically ventilated patient's response to inhaled bronchodilators. Further, it would seem reasonable to provide this therapy to only those in whom a response can be demonstrated. However, the cost-effectiveness of inhaled bronchodilator therapy is unclear, and indeed it may be less costly to provide unnecessary MDI treatments than to demonstrate that the treatments are not needed. This is particularly the case for β_2 agonists, which are not only cheap but relatively free of side effects at recommended doses. Further, as shown by Gay et al,[63] inhaled bronchodilators are likely to be beneficial in a large proportion of patients, even if a history of pulmonary disease is not present.

Some mechanically ventilated patients need no sophisticated evaluation to justify the use of inhaled bronchodilators. A patient who has previous pulmonary function tests that demonstrate reversible airway obstruction and who has used inhaled bronchodilators prior to intubation needs no further evaluation to justify use of inhaled bronchodilators during mechanical ventilation. A similar argument might be made for the mechanically ventilated patient who has not previously used inhaled bronchodilators, but has clinical findings suggestive of airflow obstruction (eg, wheezing, prolonged exhalation, smoking history). It could also be argued that a previously healthy, mechanically ventilated patient with no current or past history of obstructive lung disease does not need inhaled bronchodilator therapy (eg, trauma patient) if physical findings indicating bronchospasm (ie, wheezing) are absent.

Unfortunately, in the high-tech environment of the critical care unit, physical assessment is often overlooked or underappreciated. Auscultation is a low-cost assessment tool to evaluate bronchodilator response. An improvement in wheezing and air entry following inhaled bronchodilator administration indicates a beneficial effect.

P_{max} during volume-targeted ventilation, or V_T during pressure-targeted ventilation, may be used to evaluate bronchodilator response in some patients. These are monitoring options available on most mechanical ventilators, and require very little cost, time, or training to use. Unfortunately, P_{max} (or V_T) is a function of inspiratory resistance, and expiratory resistance may be a better indicator of bronchodilator response. Thus, there may be some patients in whom P_{max} (or V_T) is an insensitive indicator of inhaled bronchodilator response.

Evaluation of expiratory flow-volume curves, expiratory resistance, and work of breathing requires a ventilator capable of making these measurements or a commercially available lung mechanics analyzer. Systems that measure pressure and flow at the proximal airway (as well as esophageal pressures in spontaneously breathing patients) may be most useful to evaluate bronchodilator response. However, these systems are expensive ($10,000-$15,000)—probably too expensive for many community hospitals. Further, the cost of the pneumotachometer to interface the patient to these systems is $10-$25. Sophisticated measures of bronchodilator responsiveness using these devices may be more expensive than the cost of administering the MDI treatments. In other words, it may be less costly to administer some unnecessary MDI treatments to mechanically ventilated patients than to make the measurements needed to demonstrate that the treatments are not needed; as discussed previously, the cost of MDI administration in mechanically ventilated patients is relatively low (only $0.50/treatment for drugs and materials). It should be recognized, however, that lung mechanics analyzers may also be useful for evaluations other than

bronchodilator response and that this combined usefulness may justify their purchase. These devices become cost effective when their use can be demonstrated to decrease ventilator days, intensive care unit days, and hospital days. Currently few data are available that demonstrate changes in patient outcomes due to the use of these sophisticated techniques and devices.

When large doses of inhaled bronchodilator are used (eg, large numbers of actuations from the MDI, continuous or frequent treatments by SVN), more sophisticated techniques for measuring bronchodilator response are probably useful. Because large doses can be associated with undesirable side effects, it is prudent to demonstrate a beneficial physiologic response. The purchase of a lung mechanics analyzer cannot be justified solely on the basis of such treatments, however, because they are given infrequently at many hospitals. Hospitals that ventilate large numbers of patients, however, may be able to justify the purchase of such a device for a variety of reasons in addition to bronchodilator assessments. Some current-generation mechanical ventilators also have lung mechanics packages; however, few systematic evaluations of these have appeared in the peer-reviewed literature.

In Summary

Inhaled β_2 agonists can be administered by SVN or MDI during mechanical ventilation. MDI is an effective administration technique, it avoids the problems related to SVN, and it may be less expensive than SVN. A variety of techniques can be used to evaluate bronchodilator response during mechanical ventilation. Low-tech methods are also low cost, but may lack sensitivity. High-tech approaches to evaluate bronchodilator responsiveness may be more sensitive but are also more expensive. More work is needed to determine the most appropriate and cost-effective methods to evaluate bronchodilator response in mechanically ventilated patients.

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Monitoring Gas Exchange: Clinical Effectiveness and Cost Considerations

Charles G Durbin Jr MD

I. Introduction
II. General Issues Related to Monitoring
  A. Alarms as Monitors
  B. 'Routine' Monitoring
  C. Why Monitor?
  D. Monitoring Affects Cost of Care
  E. Cost Analysis of Monitoring
III. Gas Exchange Monitoring—Oxygen
  A. Pulse Oximetry
  B. Transcutaneous Oxygen Monitoring
  C. Mixed-Venous Oxygen Saturation Monitoring
  D. Arterial Oxygen Monitoring by Indwelling Sensor
IV. Gas Exchange Monitoring—Carbon Dioxide
  A. Capnometry & Capnography
  B. Transcutaneous CO_2 Monitoring
V. Clinical Efficacy, Clinical Effectiveness, and Cost-Effectiveness of Monitoring
VI. In Conclusion

Introduction

The main purposes of an intensive care unit (ICU) are to provide active therapy to very ill patients (treatment) and to watch for and prevent infrequent but important adverse events (monitoring). Continuous monitoring allows titration of therapy and identifies undesirable trends. The information that monitors provide permits timely intervention that can improve clinical outcome. ICUs emerged out of the operating and recovery rooms, where expertly trained clinicians (anesthesiologists and nurses) watch patients and administer powerful drugs to maintain unconsciousness and physiologic homeostasis. Various forms of therapy are used in ICUs to maintain physiologic stability in the face of organ system failure. In this setting, monitoring helps caregivers to maintain patients' physiologic stability and to quickly identify when therapy needs adjusting. The second goal of monitoring is the detection of undesirable trends in patient status so that appropriate interventions can be applied and a bad outcome avoided.

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One of the common meanings of "to monitor" is "to observe closely," and a monitor can be defined as an advisor, informant, or counselor. Monitors are not active devices; they do not provide patient care; they can only advise or inform those who do provide care. In evaluating the effect of monitors on patient care, it is important to remember this fact. Monitoring is only the first step in initiation of action. The person using the monitor is the essential ingredient in quality patient care. Errors in using monitors can occur because of inadequate education, inexperience, or inattention. These errors can result in an undesirable patient outcome despite adequate monitor function.

Figure 1 illustrates the process of action in an ICU. The output from a monitor suggests that further testing is needed to confirm a specific diagnosis. After these results are acquired, a diagnosis is established and an action may be taken. For example, when a ventilator monitor indicates that a low exhaled minute volume is present (monitor output), the therapist checks the circuit (additional test), and finds a partial disconnection at the endotracheal tube (diagnosis). The therapist reconnects the circuit to the patient (action), and now the exhaled minute volume monitor shows a normal value (monitor output). If the therapist had not noticed (or heard) the monitor, had not found the disconnection, or had not reconnected the patient, harm might have resulted. In evaluating the impact or value of a particular monitor, it is important to recognize the additional steps necessary to affect patient outcome—a monitoring device is no better than the person using it.

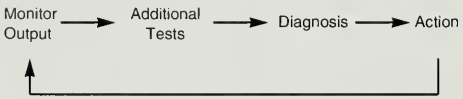

Fig. 1. Flow of information and decision making in the ICU.

General Issues Related to Monitoring

Alarms as Monitors

Sometimes in life-threatening situations, the additional diagnostic or confirmatory tests are bypassed, and action is taken without a firm diagnosis (Fig. 2). When a particular event is believed to be immediately associated with a bad outcome, action should be empiric without delaying to more firmly establish the cause. Alarms are a special feature of monitoring related to such situations. When the output variable being monitored is outside what is considered the safe range, a visual or auditory alarm is issued by the device. Once again, no change in patient care is effected by the monitoring device; the decision to act must be made by the caregiver. Caregivers often have control over the limits that trigger the monitor alarm. Given this total system, it is not reasonable to expect monitors, of themselves, to impact patient care in a positive way. Any time a monitor is evaluated for its impact on patient care, this complicated action scheme must be taken into account. Because invasive monitors always involve direct patient risks, they can easily be shown to increase morbidity. It is more difficult (if not impossible) to show that they improve patient outcome because the caregiver using the device has the major impact on outcome.

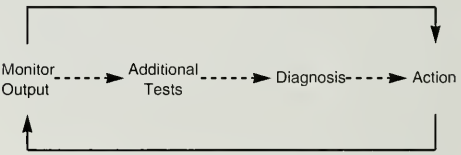

Fig. 2. When a critical value is observed, action may bypass further evaluation.

Often several monitors are used, reflecting different but related aspects of patient physiology. An experienced, watchful clinician can often respond appropriately to a critical situation without use of complicated monitors. This further confuses the objective analysis of the impact of specific monitors on patient care.

The action model shown in Figure 1 is applicable to all forms of medical diagnostic testing. The results of the first test trigger the next to confirm (or refute) a diagnosis. This sequential approach to diagnosis requires that tests be selected that are appropriate to the patient's disease process. ICU monitors should be selected for patient appropriateness as well. Patients on mechanical ventilation receiving neuromuscular blocking agents require monitoring that is different from that required by patients

breathing spontaneously without supplemental oxygen.

'Routine' Monitoring

The application of a standard battery of tests to a population of patients in an attempt to find subclinical disease is called screening. The use of screening tests may cause diagnostic problems. When screening is used indiscriminately, the likelihood of finding an abnormal result is high because of random error in the test methods or because of normal patient-population variation. The additional testing required to identify these normal 'abnormal' results increases the cost of care and may offer little or no benefit. Screening tests should be applied only when a reasonable likelihood of finding true abnormality exists. The cost-benefit of screening depends on the characteristics of the population under study. This concern has been incorporated into cost-benefit analyses of medical diagnostic tests (eg, mammography in women and prostate examination in men) with good clinical results and cost savings. It has not yet been applied to ICU monitoring but undoubtedly should be.

Monitoring in the ICU incurs a similar likelihood of finding false-abnormal results. These 'abnormals' must be worked up, wasting time and costing additional resources. This is a cost of routine monitoring. The value (benefit) and likelihood of finding a true-abnormal must be weighed against the cost of a false-abnormal finding. This concern is especially valid when one considers the alarm functions of monitors. Frequent false alarms divert the caregiver from more productive tasks and, with repeated occurrences, cause caregivers to become desensitized—to 'tune out' the sound. A true emergency may go unnoticed due to such desensitization. Evaluations of routine monitoring must include an assessment of the likelihood of a true-positive versus a false-positive alarm. The cost of monitoring includes the additional time, energy, and costs of the additional tests incurred by this problem as well as the risks incurred by frequent false alarms.

Why Monitor?

Monitors may be employed for more than a single purpose. Besides aiding the caregiver in making active interventions, monitors reduce caregivers' anxiety by confirming that the functions being monitored are within certain limits. This allows attention to be directed to other areas or to other patients. Monitors are extensions of the caregiver's senses, allowing more than one variable to be 'watched' at the same time. Table 1 lists some of the many purposes of monitors.

Table 1. Purposes of Monitoring in Intensive Care Unit Patients

Reduce uncertainty.
Reduce caregiver anxiety.
Reduce medicolegal risk.
Reduce unnecessary treatments.
Allow attention to be focused elsewhere.
Identify the need for additional therapy.
Identify undesirable trends and modify them.
Identify need for additional diagnostic studies.
Avoid lethal outcome.
Initiate action without diagnosis.
Monitor extremes.
Monitor alarm functions.
Guide ongoing therapy.
Titrate therapy.
Discontinue therapy as soon as appropriate.
Direct the use of intermittent diagnostic studies.
Confirm suspected diagnosis.
Identify false-positive findings.
Reduce number of additional intermittent tests.

The reduction in uncertainty provided by monitoring has affected medical malpractice. The widespread use of respiratory gas monitoring (pulse oximetry and capnography during surgery and anesthesia) has resulted in fewer lawsuits for poor outcome allegedly caused by inadequate ventilation and hypoxemia. Lower malpractice insurance rates have been awarded to those anesthesiologists who routinely use these forms of monitoring.[1,2] Many believe it is unlikely that the number (or kind) of adverse outcomes from anesthesia has changed greatly due to the use of monitors,[3,4] but their application is a strong defense against the claim that the cause of an adverse outcome is undetected hypoxemia.[5,6] Anesthesiologists believe that monitoring with pulse oximetry is useful even though no study has shown that improved patient outcome is attributable to its use.[7,8] The clinical detection of hypoxemia manifested as cyanosis is difficult and inexact, espe-

cially in anesthetized patients. Because irreversible harm from hypoxia occurs rapidly and laboratory evaluation of arterial oxygenation is invasive, delayed, and expensive, oximetry, with its many faults, has been accepted by clinicians who work in the operating room environment as an advance for detection of hypoxia.[9] The appropriateness of monitoring via pulse oximetry in the ICU has not been established.

Monitoring Affects Cost of Care

Monitoring for the purposes listed in Table 1 could improve the quality and reduce the cost of ICU patient care. Titration of expensive drugs to minimum effective levels and discontinuation of unnecessary treatments as soon as possible generate obvious cost savings. Avoiding organ system complications such as renal failure by minimizing toxic drug effects can save money if a prolonged hospitalization is prevented.

Some monitoring may result in increased costs. Besides the cost of the monitor itself, monitoring may lead to further testing to confirm specific diagnoses, and these additional tests add costs.

Invasive monitoring imposes additional risk to patients. The cost incurred by providing treatment for complications of invasive monitoring must be included in any assessment of the costs of such monitoring. The risks of invasive monitoring are related to the nature of the invasion and the user's technical skills and clinical experience.[10]

The choice of monitors depends on patient and clinical factors. Monitors can provide intermittent or continuous (or semicontinuous) measurements. The rate of change of the variable measured and the clinical importance of the change determines the appropriate frequency of monitoring. For the purpose of this paper, only continuous or semicontinuous monitoring is discussed. Intermittent measurements made simultaneously with blood gas analysis should be considered a type of diagnostic testing and, for the purposes of this discussion, are not monitoring. This distinction is necessary because events that adversely affect gas exchange with their consequent negative outcomes, transpire in seconds or minutes. Therefore, gas exchange monitoring for the purpose of preventing these events must be continuous.

The ideal monitor should have the characteristics listed in Table 2. Although listed first, accuracy per se is not as important as an accurate reflection of the monitored variable's changes and trends. Precision refers to the scatter around a true value. Accuracy and precision are easy to measure in controlled experiments but are less important in the clinical realm than other characteristics such as durability. Ease of use is extremely important; equipment that is complicated to set up or requires prolonged warmup time may not be used frequently or properly. Because ease of use is difficult to quantitate and because it is even harder to compare this characteristic among products, the fact that the device is actually used clinically by caregivers is evidence of that particular monitor's usefulness. Invasiveness, cost, reliability, and noise produced are other important intrinsic qualities of the devices used to monitor patients. To my knowledge, no gas exchange monitor is yet available that incorporates all of the desired characteristics listed in Table 2.

Table 2. Characteristics of the Ideal Monitor

Accurate	Noninvasive
Precise	Inexpensive
Reliable	Safe
Easy to use	Compact
Easy to repair	Quiet
Meaningful	Indestructible
Continuous	Portable

Cost Analysis of Monitoring

Complete cost accounting of monitoring is difficult because the value of associated variables, such as additional diagnostic tests required and the benefits accrued due to prevention of costly complications, cannot be fully known. However, the following basic approach can be used in considering this problem.[11-13] Direct costs of monitoring include

- capital—the cost of the equipment, service, and replacement,
- labor—the cost of personnel to run and maintain the equipment, the education needed to maintain proficiency in its use, and

- materials—the reagents and disposable components needed for each patient use.

To these must be added the indirect costs:

- additional testing costs needed to respond to a monitor output,
- treatment costs added by monitoring including costs of complications from monitoring devices, and
- treatments related to correcting the identified abnormalities.

Cost savings may also occur and should be considered in the cost analysis of a particular monitor, including

- cost of tests replaced by the monitor output,
- savings from decreased hospital stay, and
- savings realized by the prevention of more costly treatments and complications.

Potential savings are difficult to estimate, but should be subtracted from the total cost when a complete cost analysis of monitoring is performed (Table 3).

Table 3. Components of a Cost Analysis

Direct Costs
Capital
Labor
Materials
Indirect Costs and Savings
Tests added
Tests avoided
Treatment costs added
Treatment costs avoided
Caregiver Impact
Apparatus attention (saving if others do it)
Apparatus attention (cost if it adds work)
Additional tasks (phone calls, time to perform additional tests)
Tasks reduced
Outcome Benefits
Increase in quality of life
Decrease in morbidity
Improved safety
Other patients may receive more attention
Side Effects, Risks

Monitors can affect caregiver efficiency in activities other than laboratory testing. If the device is user-friendly, needs little attention, and provides important and useful information accurately, time and energy for other important tasks are made available and may be counted as a cost reduction. If the device requires frequent attention, provides poor quality or redundant information, or necessitates additional time-consuming confirmatory testing, the caregiver may be distracted from the more important task of treating the patient. Patient risk and potential benefit from monitoring are difficult to quantitate and evaluate.

Table 4. Monitors of Gas Delivery and Exchange

Oxygenation
Bulk O_2 tank analysis, fill level
Piped-oxygen pressure monitor (alarm)
Ventilator blender pressure alarm
Inspired O_2 analyzer
Intermittent ABG
Transcutaneous O_2 monitor
Pulse oximeter
Continuous ABG
$S_{\bar{v}O_2}$ monitor
Shunt measurement
Exhaled gas analysis ($\dot{V}_{O_2}$)
Tissue oxygen electrode
Ventilation
Airway pressure
Inhaled, exhaled, and minute volumes
Gas flow
Compliance
Work of breathing
Exhaled gas analysis
End-tidal CO_2
Intermittent ABG
Transcutaneous CO_2
Continuous ABG

Many components of the gas supply and/or ventilator system that can affect gas exchange can be monitored. Some of these are listed in Table 4. With the cost analysis framework in mind, this paper is limited to continuous and semicontinuous monitoring of respiratory gas exchange. Only a quasi-comparative analysis is possible because many of the cost-benefit details of monitoring have not been determined.

Gas Exchange Monitoring—Oxygen

The flow of oxygen from the bulk source to the mitochondrion is illustrated in Figure 3. When mitochondrial oxygen depletion occurs it may be due to a failure at any of these steps. Possible mechanisms for failure at these points and representative types of monitoring for some of these steps are listed in Table 5. Some monitors are specific and diagnostic because they reflect only one isolated aspect of the process. For example, if the bulk oxygen system is exhausted, the empty-tank alarm indicates precisely what is wrong. The line-pressure monitor also detects this problem but several other failures could also cause low line pressure. If an upstream failure (eg, piped gas failure) continues for several minutes, the downstream monitor (eg, oximetry or continuous blood gas analysis) reflects the patient's response to this failure but is not diagnostic of the specific problem. Rapid and precise location of the problem is more likely when several different monitors are employed at different critical steps.

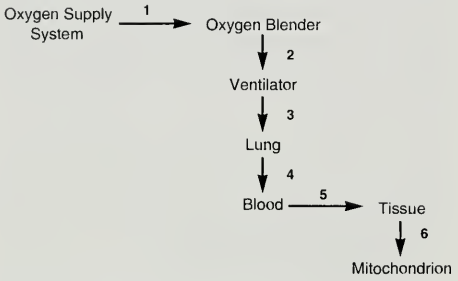

Fig. 3. The flow of oxygen from the supply system to the mitcochondrion.

Currently, there are four continuous blood oxygen monitoring systems available: pulse oximeters, transcutaneous oxygen monitors, continuous mixed-venous oxygen saturation monitors (Swan-Ganz catheters), and continuous indwelling arterial oxygen sensors. Each can be used to titrate therapy and detect oxygenation failure from upstream or down-

Table 5. Oxygenation Failure: Possible Mechanisms and Monitors

Location of Failure	Possible Causes of Failure	Possible Monitors of Delivery Step
Supply system	Empty tank	Tank gauge
	Damage to pipelines	Line pressure gauge
	Demand system overload	System flow gauge
Blender	Malfunction	Oxygen sensor, gas analysis
	Improper O_2 setting	Oxygen sensor, gas analysis
	Improper gas	Pin index system, gas ganalysis
Ventilator	Incorrect O_2 setting	Oxygen sensor, gas analysis
	Mixing valve malfunction	Oxygen sensor, gas analysis
Circuit	Disconnection	Low pressure alarm
		Low minute ventilation alarm
		Apnea alarm
Endotracheal tube	Extubation	Low pressure alarm, Apnea alarm
	Cuff leak	Low minute ventilation, end-tidal CO_2
	Endobronchial intubation	High pressure alarm, compliance monitor
	Occluded tube	High pressure alarm, compliance monitor
Lung	Hypoventilation	Low minute ventilation, end-tidal CO_2
	Shunt effect	Pulse oximetry, continuous ABG's
	Bronchospasm	High pressure alarm, compliance monitor
Arterial blood	Decreased cardiac output	End-tidal CO_2, $S_{\bar{v}O_2}$ monitoring
	Hemoglobinopathy (ie, COHb)	Hemoglobin spectroscopy (intermittent)
	Hypoxemia	Pulse oximetry
Tissue	Poor perfusion	Transcutaneous O_2 monitor
	Hypoxemia	Pulse oximetry
	Uptake failure (ie, cyanide)	$S_{\bar{v}O_2}$ monitoring, tissue oxygen monitors, magnetic resonance imaging

stream delivery failures. Each of these has its own characteristics, optimal uses, risks, and problems. These issues and the cost-benefit of each of these are analyzed in detail later in this paper.

Pulse Oximetry

Although no published or agreed-upon standard exists (outside of the operating theater), pulse oximetry monitoring is widely practiced in patients receiving mechanical ventilation in ICUs.[14-18] Pulse oximetry is based on the differential absorption of light by oxygenated and reduced hemoglobin (Hb) in blood, and the changes in absorbance coincident with arterial pulsation. The accuracy of these devices has been extensively evaluated.[15-17] Pulse oximeters have an acceptable accuracy in critically ill patients as long as arterial oxygen saturation exceeds 75%.[16] At lower values, differences between CO-oximeter and pulse oximetry saturations (S_{aO_2} and S_{pO_2}, respectively) may exceed 10%.[17] Oximetry inaccuracies have been noted at $S_{pO_2} \leq 90\%$ in patients with heavy skin pigmentation.[18] As a result, critical S_{pO_2} values of 92% for White patients and 95% for Black patients have been suggested. Some dyes significantly alter the accuracy of pulse oximetry;[19] however, bilirubin does not appear to affect oximetry accuracy.[20,21] It is important to remember that the relationship between saturation and partial pressure is affected by many factors. S_{aO_2} is not identical to P_{aO_2} and, in fact, the relationship between the two is rarely linear. Another factor reducing accuracy of pulse oximeters is blood flow through the point of attachment (digit or ear).[22-30] Hypoperfusion may result from hypotension, hypothermia, congestive heart failure, or the administration of vasoconstrictive drugs. The presence of carboxyhemoglobin or methemoglobin may produce a falsely elevated S_{pO_2}.[23,24] Despite these limitations, pulse oximetry yields useful information, especially about trends in oxygenation. Its ability to function in the clinical environment and provide meaningful data has been tested. Its routine use has not been shown to improve patient outcome. Oximetry increases caregiver comfort (reduces uncertainty) and may reduce medicolegal risk (cost).

In a large prospective, randomized study reported by Moller and colleagues,[7,8] hypoxemia, hypoventilation, and endobronchial intubation were identified more frequently and quickly in patients continuously monitored with oximetry during the perioperative period. These monitored patients also remained longer in the recovery room, received more invasive blood gas determinations, and received more supplemental oxygen than those who were not monitored by pulse oximetry. The incidence of myocardial ischemia (identified on ECG) was significantly less in the group with pulse oximetry monitoring. However, ultimate outcome variables (mortality and cardiovascular morbidity) did not differ significantly between the groups. In this study, the failure rate of oximetry was significant. This was especially important in the high-risk-patient group in which 7% of patients were not successfully monitored for a significant period of time. Despite failure of the study to show improved outcome, the anesthesiologists in the study said they would use oximetry routinely in their practice.

The routine use of pulse oximetry on all patients in the ICU is probably not clinically useful or cost-effective due to normal variations of S_{pO_2} in stable patients that is interpreted as an abnormal test requiring confirmation by arterial blood gas (ABG) analysis. However, use of this technology in high-risk patients, such as those on high levels of ventilatory support, and especially those receiving neuromuscular blocking agents, is justified and probably cost-effective. Unfortunately, these are the patients in whom the monitor is likely to fail to provide an adequate signal.[25] Due to its noninvasive character, direct patient risk from oximetry is almost nil in adults and children. (Burn injuries and pressure necrosis have been reported in neonates.[26,27]) The device works immediately, requires little attention, and is reasonably durable. For these reasons, this is a popular monitor among caregivers. Devices that provide a plethysmographic pulse tracing or indicator to help distinguish electrical interference from a true signal and thereby improve interpretation are preferred over those producing only a numerical output.[14,28]

Transcutaneous Oxygen Monitoring

Transcutaneous oxygen tension (P_{tcO_2}) is measured with heated electrochemical sensors. The amount of current transmitted between a platinum or gold cathode and a silver anode is directly pro-

portional to the P_{O_2} at the cathode surface.[29] The sensor is usually applied to the trunk in critically ill patients because limb perfusion varies considerably. In adult patients, the sensor should be applied to the skin and heated to 44° C for at least 20 minutes before the validity of the measurement can be assessed. The heated sensor encourages vasodilation and improves the agreement between arterial blood and transcutaneous oxygen tensions. Heating the skin reduces the influence of local perfusion on the measurement. However, the heating process cannot remove the influence of tissue edema or skin thickness on the measurements. Technical difficulties with transcutaneous sensors include

- the need to rotate sites every 2-4 hours to avoid skin burns,
- careful skin and electrode preparation, and
- lengthy calibration times.[30]

P_{tcO_2} values are generally lower than P_{aO_2} values. Some investigators believe that P_{tcO_2} more closely reflects tissue oxygen tension.[31,32] In normal subjects, the gradient between P_{aO_2} and P_{tcO_2} ($P_{tc} - P_{aO_2}$) is stable. However, in critically ill patients, $P_{tc} - P_{aO_2}$ is highly variable.[31,32] This is particularly true in patients with reduced cardiac index (CI).[33] Hasibeder et al[34] evaluated the influence of P_{aO_2}, mean arterial pressure (MAP), CI, oxygen dissociation curve, and Hb on P_{tcO_2} values. Significant linear correlations of P_{tcO_2} with P_{aO_2} and MAP were found, with r values of 0.6 and 0.42, respectively. However, only 40% of the variability in P_{tcO_2} could be explained by P_{aO_2} and MAP. They concluded that the hemodynamic, respiratory, and local influences affecting P_{tcO_2} measurements are poorly understood in critically ill patients.

Trending P_{tcO_2} values may be useful in the critically ill patient. Reductions in P_{tcO_2} values not associated with changes in P_{aO_2} imply reductions in local perfusion of skin tissue. P_{tcO_2}-P_{aO_2} ratios have been correlated with CI.[35] Hence, bedside trending of P_{tcO_2} may alert the clinician to hemodynamic alterations, even when P_{aO_2} is stable. P_{tcO_2} measurements have been used to rapidly titrate PEEP to an optimal level.[36] The effects of increasing PEEP on improving arterial oxygenation and decreasing cardiac output (C.O.) may be weighed by observations of changes in the P_{tcO_2} value.

This monitor is used less frequently in adults than in infants and children. The device is bulky, delicate, expensive, requires frequent maintenance, and takes a long time to warm up. There is a moderate degree of patient risk; burns have been reported. The need to change the sensor site every 2 or 3 hours and the difficulty interpreting the output value reduce caregiver interest in routinely using this monitor. Selective application of this technology (eg, weaning from mechanical ventilation) may be useful and cost-effective.

Mixed-Venous Oxygen Saturation Monitoring

Special pulmonary artery catheters (PACs) permit continuous display of mixed-venous oxygen saturation, $S_{\bar{v}O_2}$. Embedded in the wall of the catheter are fiberoptic filaments that transmit and detect light reflected from Hb. By comparing the reflectance at several wavelengths, computer analysis permits calculation and display of $S_{\bar{v}O_2}$.[37] Intermittent measurements of $S_{\bar{v}O_2}$ can be obtained from CO-oximeter analysis of blood aspirated from the PAC for calibration and comparison. Values for $S_{\bar{v}O_2}$ as determined by reflectance oximetry correlate well with CO-oximeter measurements, provided the catheter tip is not abutting a vessel wall or occluded by clot or debris.[38]

Mixed-venous oxygen saturation reflects the balance between oxygen consumption ($\dot{V}_{O_2}$) and delivery, and normally ranges from 70-75%.[39] $S_{\bar{v}O_2}$ is influenced by S_{aO_2}, CI, Hb, and oxygen consumption ($\dot{V}_{O_2}$). When interpreting $S_{\bar{v}O_2}$ changes, these variables should be considered.

Mixed-venous oxygen saturations below 65% suggest a poor reflectance signal, inadequate oxygen delivery, or excessive $\dot{V}_{O_2}$. Reduced oxygen delivery may result from reduced CI, S_{aO_2}, or Hb. Documentation of a falling $S_{\bar{v}O_2}$ mandates evaluation of each of these as potential causes. Therapy should then be directed at correcting the identified cause(s) of the change in $S_{\bar{v}O_2}$. Excessive $\dot{V}_{O_2}$ is known to accompany increased muscular activity (ie, shivering, seizures), fever, malignant hyperthermia,[40,41] and the sepsis syndrome.

Spuriously elevated venous oxygen saturation readings may be caused by persistent wedging of

the pulmonary artery catheter (thus measuring saturated pulmonary capillary blood), increased oxygen delivery, or reduced $\dot{V}_{O_2}$. Increased oxygen delivery may be seen in patients with peripheral shunts, sepsis, allergic reactions, or intracardiac shunts. Reductions in $\dot{V}_{O_2}$ and a rise in $S_{\bar{v}O_2}$ are seen when peripheral oxygen uptake is decreased due to toxins such as cyanide.[42] The interpretation of trends in $S_{\bar{v}O_2}$ requires additional testing. (The $S_{\bar{v}O_2}$ may be correlated with outcome, eg, patients with low $S_{\bar{v}O_2}$ after CPR and myocardial infarction do poorly.[43,44])

Cost-effective analyses of this device have been reported.[45-47] The additional cost of the catheter (above the usual PAC) is approximately $100. Savings may occur from a reduced number of ABG analyses. Venous blood gas analyses may be increased because calibration samples are needed daily. With the now frequent use of pulse oximetry, the savings attributable to $S_{\bar{v}O_2}$ monitoring is probably less than originally estimated. The use of these two monitors together provides rapid determination of which of the four variables (Hb, Cl, S_{aO_2}, or $\dot{V}_{O_2}$) is responsible for a change in $S_{\bar{v}O_2}$. This form of monitoring is called dual-oximetry and can also be used continuously to reflect intrapulmonary venous admixture (shunt).[46] A reduced number of C.O. measurements may result. This saves the costs associated with supplies and caregiver time, and may result in weaning of the patient from hemodynamic and ventilatory support more rapidly.[47] The major value of this monitor seems to be that of an early warning system for imbalance of oxygen supply and demand.[48] It is not specific for a particular change; however, in controlled circumstances, it can be used as a surrogate measurement of changes in C.O., P_{aO_2}, or $\dot{V}_{O_2}$.

Arterial Oxygenation Monitoring by Indwelling Sensor

The newest monitor of oxygenation is based on indwelling electrochemical sensor technology. The fluorescent optode continuously measures arterial oxygen partial pressure (P_{oO_2}), pH, and carbon dioxide partial pressure (P_{oCO_2}) through a small filament inserted through an arterial catheter.[49] A miniaturized Clark electrode device to measure P_{aO_2} is in use in Europe.[50] Usual pressure-monitoring and blood-drawing activities are unaffected by the devices. The devices repeatedly measure the variables and update values continuously. There is a sensor lag time of about 2 minutes (for equilibration) with the optode system.[51] Accuracy of these devices is good over the clinical range of the measurement.[52]

Clinical performance of the optode device appears promising. In a case report by Greenblott and colleagues,[53] pulse oximetry, transcutaneous monitoring, and a three-component fiberoptic optode (Cardiovascular Devices Inc, Irvine CA) were used during one-lung anesthesia. The optode device functioned well and was more effective at predicting actual ABG values than the other devices. Pulse oximetry failed at several critical times during the procedure and was not useful at all when the P_{aO_2} exceeded 100 torr (as saturation remained 100%). Figure 4 summarizes the changes in oxygenation reflected by these monitors during the case. P_{oO_2} reflected wide changes and accurately predicted ABG values, P_{tcO_2} averaged the peaks and valleys, and S_{pO_2} failed to report useful values during critical events throughout the case. More experience is needed to assess the place of this new technology in clinical care.

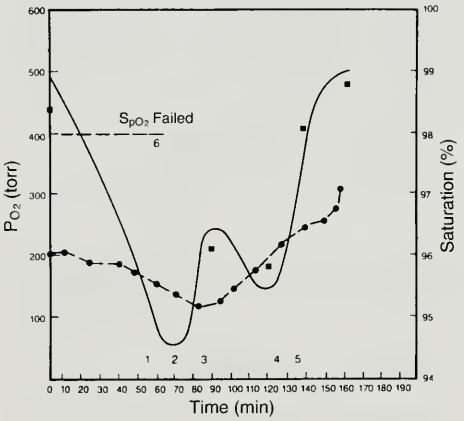

Fig. 4. A comparison of several monitors of oxygen during anesthesia for pulmonary resection. P_{aO_2} (ABG) = ■, P_{tcO_2} = ●–●–●, S_{pO_2} = – – –, P_{oO_2} = ——. (Reprinted from Reference 53, with permission.)

Optode devices are expensive, require considerable user attention, and are fragile. Their individual patient-use cost is high. However, they provide in-

formation that is not available continuously from any other monitor. Currently, the sensors are used for only 72 hours or less. Once arterial correlation has been established, the use of the optode device should decrease the number of ABG analyses needed. None of the other oxygen monitors have this potential. The alleged improvements to patient care by this monitor remain to be shown.[54,55] Reliability, durability, and ease of use have yet to be demonstrated.

Gas Exchange Monitors—Carbon Dioxide

The other component of gas exchange is CO_2 elimination. CO_2 is the end product of oxygen metabolism. Approximately 70,000 mEq of CO_2 are excreted by the lungs each day. The steps in CO_2 elimination are illustrated in Figure 5. Major interference with CO_2 excretion occurs with decreased C.O. (delivery to the lungs), increased physiologic dead space (eg, pulmonary embolism, COPD), or ventilatory failure. CO_2 may be monitored continuously by capnography of exhaled gases, transcutaneous CO_2 monitoring, and continuous indwelling arterial CO_2 monitoring (P_{oCO_2}). Although the flow of CO_2 is through the venous blood to the lungs, changes in CO_2 delivery are usually reflected in arterial P_{aCO_2}, because the venous-to-arterial gradient for CO_2 is very small.

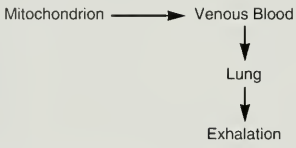

Fig. 5. The mass movement of carbon dioxide from the tissues to the room.

Capnometry & Capnography

The measurement of CO_2 concentrations in expired gases (end-tidal CO_2, P_{etCO_2}) is mandatory in operating rooms and is frequently used in ICUs. Bedside monitoring devices incorporate infrared light absorption, Raman light-spectrum scattering, or mass spectroscopy to estimate CO_2. The two types of capnometers are mainstream (the sensor is in-line with the breathing circuit) and sidestream (part of the exhaled gas is withdrawn from the patient circuit and delivered to the monitor). Mechanical problems with both types of sampling systems can result in over- and underestimation of the CO_2 fraction.[23]

Errors with mainstream sensors generally result from mucus or liquid obstruction of the light path. Sidestream units may provide erroneous data when mucus or moisture collects in the sampling tubing or when the tubing becomes kinked. When sampling rates are too high, room air may be entrained through the sampling catheter and dilute exhaled gas.[56]

Capnometry and capnography are useful for confirmation of tracheal vs esophageal intubation, identification of dislodgment of an endotracheal tube, and ventilator malfunction or disconnection.[57-60] Capnography can be used to estimate P_{aCO_2} in some situations. P_{aCO_2} is often equated to P_{etCO_2}. However, many factors affect P_{etCO_2} other than P_{aCO_2}. Increases in CO_2 production ($\dot{V}_{CO_2}$), increased pulmonary perfusion, and acute alveolar hypoventilation result in increased P_{etCO_2}, yet may not perceptibly change P_{aCO_2}.[61,62] The arterial-to-end-tidal gradient ($P_{aCO_2} - P_{etCO_2}$) varies under these and other circumstances and, therefore, the value of P_{etCO_2} in predicting the actual P_{aCO_2} is limited. Negative arterial-to-end-tidal gradients may be seen in 12% of normal subjects undergoing anesthesia who are ventilated with large tidal volumes at low frequencies.[63] Negative gradients are also seen in 50% of pregnant subjects,[64] 8% of patients following cardiac bypass surgery,[65] and 50% of infants.[66] Caution should be used when interpreting P_{etCO_2} as an estimate of P_{aCO_2}. Steady state conditions are necessary for this assumption to hold.

Changes in P_{etCO_2} and the capnogram may be of clinical value. Abrupt reductions in P_{etCO_2} that occur when the ventilator circuit is intact and the endotracheal tube remains in the trachea can indicate pulmonary embolism.[67] P_{etCO_2} correlates well with measured C.O. during resuscitation and can be used to gauge the effectiveness of cardiac compressions.[68,69] The $P_{aCO_2} - P_{etCO_2}$ gradient also serves as an estimate of physiologic dead space and can be used as a guide to achieving "best-PEEP."[70]

Capnographs vary in cost depending on the method used to determine P_{etCO_2}. Disposable supplies are frequently expensive and may not be us-

able beyond several hours. Capnometry must be frequently correlated with ABG values to interpret changes, thus increasing cost. In our experience, mass spectrometers are large and delicate and may often require sophisticated service and frequent adjustments. They require external calibration with expensive standards. Use of capnometry during weaning trials, during optimization of ventilatory support with reduced intracranial compliance, and for ventilator disconnection detection in very critical patients (ie, patients receiving neuromuscular blocking agents) is helpful and may be cost-effective. CO_2 detection is the 'gold standard' for confirming tracheal intubation. However, I believe that continuous monitoring of P_{etCO_2} has yet to find its place in critical care.

Transcutaneous CO_2 Monitoring

Transcutaneous sensors, similar in design to those used to transcutaneously measure oxygen, are used to measure CO_2 (P_{tcCO_2}), and the technical concerns are similar for both. Often, the two sensors are combined within a single probe. As a result of skin heating, a local increase in local $\dot{V}_{CO_2}$ may occur, and values for P_{tcCO_2} often exceed P_{aCO_2}.[71] P_{tcCO_2} measurements remain linearly correlated with P_{aCO_2} values even in critically ill patients ($r = 0.76$) because they are less affected by changes in cardiac function and acid-base status than are P_{tcO_2} measurements.[34] This is partly because of the small arterial-to-venous P_{CO_2} difference normally present. Acute changes in CO_2 excretion are not reflected rapidly by transcutaneous monitors. Because of this delay, P_{tcCO_2} is a poor detector of circuit disconnection or apnea.

As with P_{tcO_2} monitoring, difficulties with these devices include the time necessary for calibration and stabilization (less of a problem for P_{tcCO_2} than for P_{tcO_2}), the need to frequently change monitoring site, delicacy of the device, and the large size of the probe (particularly important for infants and children). I have found them to be expensive and finicky and to require a great deal of user attention. I believe that the clinical effectiveness of these devices in adults is minimal. In adults, selective use in some carefully controlled situations may be cost-effective because they reduce the need for other tests.

CO_2 Monitoring by Indwelling Sensor

Optode technology permits arterial CO_2 monitoring (P_{oCO_2}) in combination with pH and P_{oO_2} monitoring directly through an arterial catheter. As described earlier, this technology is accurate and precise and requires a short warmup period. It is, however, expensive and delicate. The use of this device may reduce the need for other ABG analyses. It has not yet been determined what types of patients benefit from monitoring with this device.

Clinical Efficacy, Clinical Effectiveness, and Cost-Effectiveness

Clinical efficacy is the ability of a device or treatment to achieve its ascribed goals under controlled circumstances.[72] Clinical efficacy of a monitor means that its output consistently correlates with the value of a physiologic entity and/or with other monitors whose accuracy is established and accepted. Clinical efficacy also means that the monitor contributes to a desirable patient outcome with a known disease process in prospective, controlled studies that have been performed in carefully selected clinical situations. Some monitors have been tested in this way and have demonstrated accuracy, predictability, and correlation with expected outcome.

Clinical effectiveness, on the other hand, is the ability of the device or treatment to achieve the same desired results during less rigorously controlled use,[73] and in general, this quality has not been demonstrated for any blood-gas monitoring device.

Clinical effectiveness should reflect (among other things) the balance between accuracy, ease of use, and production of meaningful information. A monitor will be used only

- if its use is mandated by policy, and/or
- it is felt to be 'worth the trouble' by the user (if given a choice).

A monitor or monitoring is cost-effective when, in addition to being clinically effective (or at least useful), it costs less or incurs less risk than other accepted monitors currently in use. New (or existing) technology should not be introduced unless it is

- less costly and at least as effective as the current standard,
- more costly but more effective, and its added benefit is worth the added cost, or
- less effective and less costly but the benefit of the current standard is not worth the added cost.[74-76]

Most of the monitors described in this paper can be used in a cost-effective fashion. A comparison of relative costs and benefits is provided in Table 6.

Pulse oximetry is useful in high-risk patients because of its specificity for hypoxemia, low cost/patient, ease of use, and negligible patient risk. In certain clinical settings (diagnosing the absence of hypoxemia), this monitor can replace the need for intermittent ABG analysis. However, indiscriminate use of pulse oximetry may lead to an increase in intermittent ABG analysis. The cost-effectiveness of this monitor depends on careful patient selection.

$S_{\bar{v}O_2}$ monitoring has been shown to reduce the number of venous blood gas analyses and C.O. determinations required in critically ill patients on vasopressors.[53] However, this savings only accrues in patients who would have had these studies performed without the $S_{\bar{v}O_2}$ catheter. Generally, there are only a few such patients in the ICU—those who are most unstable and those requiring active therapy titration on a minute-to-minute basis.

Similar statements may be made about transcutaneous blood gas and end-tidal CO_2 monitoring. With careful patient selection, a reduction in ABG analyses and other tests used for monitoring may generate cost savings. If used indiscriminately, however, the cost of care may increase.

There are other techniques that may reduce the use of 'routine' blood analyses obtained for monitoring oxygenation and ventilation in stable patients. Simply removing the indwelling arterial catheter remarkably reduces the number of ABG analyses performed, with no demonstrable effect on quality of care.[77] Use of an algorithm to identify clinical indications and for education of ICU staff in application of these indications is also effective.[77,78]

Indwelling ABG devices may have a cost benefit. This is because they eliminate the need for additional ABG analysis except those infrequently required for calibration. Unlike the other monitors of gas exchange, these devices measure the actual physiology of interest instead of a surrogate or corollary. Changes in the monitor's output that call for patient intervention need not be confirmed by ABG analysis prior to caregiver response. Device malfunction rate, impingement on a vessel wall, drift in calibration, and delay in demonstrating changes have not been evaluated under less than controlled conditions. Thus, the clinical efficacy has been demonstrated but I believe that the clinical effectiveness is yet to be proved. Caregivers must have confidence in the monitor's output to abandon use of confirmatory ABG analysis when this device is used. The development of such confidence takes time, and cost savings with early or infrequent use are unlikely. In evaluating the output from the other monitors, confirmatory ABG analysis is usually mandated.

It remains to be seen whether any continuous monitor of gas exchange improves patient outcome.

Table 6. Comparison of Relative Costs and Benefits among the Available Continuous Gas Exchange Monitors

Device	Direct Costs			Indirect Costs	Benefits	Patient	Clinical
	Capital ($)	Labor	Materials ($)	Added Tests	Reduced Tests	Risks	Utility
Pulse oximeter	5,000	Low	5-50	Moderate	High*	Low	High
Transcutaneous†	20,000	High	50-100	High	Low	Moderate	Low
Mixed venous saturation (PAC)	25,000	Low	150	High	Moderate	High*	Moderate
Continuous ABG†	30,000	Moderate	200	Low	High‡	Moderate	Moderate*
Capnography	15,000	High	50	Moderate	Moderate	Low	Moderate

*Presumed but not demonstrated.
†CO_2 and O_2 monitoring.
‡If Swan-Ganz catheter inserted for this purpose only.

I believe that the use of these technologies is now based on inadequate clinical evidence of benefit and operator uncertainty. The reason for employing these expensive devices is to better care for patients who are at high risk for mortality or morbidity and who are clinically unstable. The possibility of reducing other costs with use of these monitors should be considered. Cost-effective use requires considerable judgment in application.

In Conclusion

Monitors, of themselves, never improve patient outcome because they do not do anything. They provide information that must be interpreted. The decision to act (or not act) must be made by a person. The value of monitoring must be interpreted in light of this fact. Monitors do impose risks and cost money. The analysis of outcome of monitoring is likely to show that patient morbidity is directly related to the invasiveness of the monitor. Not all risks associated with monitors are due to mechanical factors. If a device's output conveys a false sense of security to caregivers when the patient is really in trouble, monitoring can add to patient morbidity and risk. If the caregiver pays too much heed to a monitor's output and fails to watch the patient directly, harm can result. Monitoring increases cost of care. The usefulness, effectiveness, and value of most monitors have not been convincingly demonstrated. The use of certain monitors may be considered a standard of care but may, in fact, be dictated by unsupported opinion based on theoretical models of pathophysiology. Often these patterns, supported only by anecdotal experiences of caregivers, are codified into recommendations, guidelines, and policies.

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Flow-Triggering Systems

Richard D Branson RRT

Introduction

According to Chatburn,[1] a breath can be described by defining its control and phase variables—the trigger, limit, and cycle variables.

The trigger variable can be time, pressure, volume, or flow. Time-triggering occurs when the ventilator delivers a breath according to the set frequency, independent of the patient's spontaneous effort. Time-triggering always results in the delivery of a preset number of mandatory breaths. Pressure-, volume-, and flow-triggering occur when the patient's effort causes the set sensitivity to be breached and may result in delivery of a mandatory or a spontaneous breath (Fig. 1). More simply, these may be called patient-triggered breaths and specification of the variable used for triggering reserved for more technical discussions. A ventilator can also have a mandatory, or manual, trigger that begins inspiration when a button is depressed.

The trigger variable and its influence on the imposed work of breathing (WOB_I) have been a subject of considerable scientific scrutiny.[2-10] These studies compare the ability of various ventilators to respond to simulated spontaneous breathing and report the WOB_I or some other inference of patient work (eg, peak negative pressure).

Prior to 1988, the majority of ventilators were capable of only time-, mandatory, and pressure-triggering. Flow-triggering was available on the Engstrom Edith ventilator, but to my knowledge was not widely used or studied. Flow-triggering was reintroduced by Puritan-Bennett in the late 1980s as "Flow-By." Interestingly enough, Flow-By was developed as a method of allowing simulated T-piece weaning while eliminating the need for a separate aerosol setup and preserving the monitoring capabilities of the ventilator; however, recent work has shown flow-triggering to be superior to pressure-triggering in laboratory and clinical studies.[2-10]

In this Corner, I describe the pressure- and flow-triggering methods used by adult mechanical ventilators capable of both flow- and pressure-triggering.

Ventilator Descriptions

Bird 8400STi

During pressure-triggering, the Bird 8400STi is pressure-triggered (sensitivity is adjustable from –1 to –20 cm H_2O below baseline), pressure-limited, and pressure-cycled. When the sensitivity threshold is reached, flow up to 120 L/min is delivered in an attempt to maintain pressure at set baseline pressure (pressure limit). Inspiration is cycled when pressure rises 1.0 cm H_2O above baseline (pressure-cycled). Airway pressure is measured by a pressure transducer on the expiratory side of the ventilator.

During flow-triggering (called flow support), the 8400STi is flow-triggered, flow-limited, and flow-cycled. This is unique to the 8400STi because most flow-triggering systems use pressure as the limit variable. A 10-L/min baseline flow (nonadjustable) is activated when flow support is selected. A flow-trigger of 1-10 L/min is selected by the operator. The 8400STi compares flow passing through the inspiratory and expiratory flow transducers; if flow sensitivity is set at 2 L/min, then inspiration is triggered when flow through the expiratory transducer falls below 8 L/min. The 8400STi provides sufficient inspiratory flow to maintain flow exiting the expiratory flow transducer at 10 L/min (flow-lim-

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The information provided here was taken from the respective operator manuals. A product manager for each manufacturer was contacted when necessary to provide additional information and to verify the content. Each manufacturer has reviewed the content relevant to its ventilators. The author has no financial interest in any of the products described.

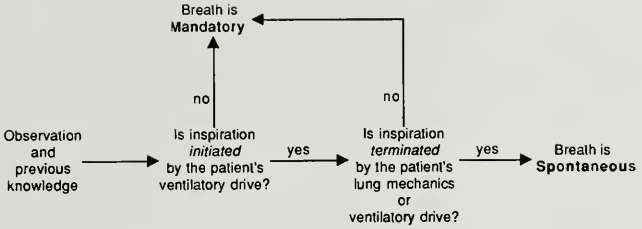

Fig. 1. Algorithm defining spontaneous and mandatory breaths. If the breath is *triggered* according to a preset frequency or minimum minute ventilation or *cycled* according to a preset frequency or tidal volume, the breath is mandatory. All other breaths are *spontaneous*. (Reprinted from Reference 1.)

ited). During inspiration, a flow up to 120 L/min is provided. When the baseline flow returns to 10 L/min and the expiratory flow transducer measures 10 L/min, inspiration is flow-cycled.

If flow-triggering fails to function (eg, the flow transducer is damaged), a secondary pressure-trigger is activated at –5 cm H_2O. If the pressure-trigger is activated when flow support has been selected, the sensitivity display flashes.

Hamilton Veolar FT

During pressure-triggering, the Hamilton Veolar FT is pressure-triggered (adjustable from –0.5 to –10.0 cm H_2O), pressure-limited (1.5 cm H_2O above PEEP), and flow-cycled (inspiration ends when flow decays to 12-37% of initial peak flow). Pressure and flow are measured at the inspiratory outlet of the ventilator.

During flow-triggering, the flow sensitivity is set by the operator at 3-15 L/min. The baseline flow is then automatically set at twice that value (6-30 L/min). When inspiratory flow across the proximal flow transducer exceeds the trigger sensitivity, flow up to 188 L/min is provided in an attempt to maintain a pressure 1.5 cm H_2O above baseline pressure. Inspiration is cycled off when flow decays to 12-37% of initial peak flow. During flow-triggering, the Veolar measures flow for triggering and cycling at the proximal airway, and pressure is measured in the inspiratory limb of the machine.

In the event of a failure of the flow sensor, pressure-triggering occurs automatically at –3 cm H_2O.

Puritan-Bennett 7200ae

During pressure-triggering, the 7200ae is pressure-triggered (adjustable from –0.5 to –20.0 cm H_2O) and pressure-limited. When the sensitivity threshold is reached, the 7200ae delivers flow up to 180 L/min in an attempt to maintain pressure (pressure limit) at baseline pressure minus sensitivity. For instance, if PEEP is 10 cm H_2O and pressure sensitivity is –2 cm H_2O, then the 7200ae will maintain a pressure of 8.0 cm H_2O throughout inspiration. Inspiration is pressure-cycled when airway pressure rises 1.0 cm H_2O above baseline pressure, signaling patient expiratory effort.

During flow-triggering, the 7200ae allows the clinician to set a baseline flow of 5-20 L/min and a flow-trigger sensitivity of 1-10 L/min provided the flow sensitivity is not greater than one half the baseline flow. Inspiration is flow-triggered when the expiratory flow transducer measures a diminution in flow (compared to the flow sensed by the inspiratory flow transducer) equivalent to the flow sensitivity setting. Inspiration is then pressure-limited at 0.5 cm H_2O above the baseline pressure. Inspiratory flow up to 180 L/min is provided. Inspiration is flow-cycled when flow through the expiratory flow transducer exceeds flow through the inspiratory flow transducer by 2 L/min.

Siemens Servo 300

During pressure-triggering, the Siemens 300 allows sensitivity to be set from 0 to –17 cm H_2O. Inspiratory flow is available up to 180 L/min in the adult mode, 30 L/min in the pediatric mode, and 12 L/min in the neonatal mode, and is pressure-limited at the set-PEEP. Inspiration is flow-cycled when flow decays below 5% of the peak inspiratory flow or after 80% of the Breath Cycle Time setting has elapsed. As a secondary cycle, inspiration is cycled when pressure exceeds the preset pressure-control setting above PEEP. During pressure-triggering, the ventilator provides a constant bias flow (2 L/min in the adult mode, 1 L/min in the pediatric mode, and 0.5 L/min in the neonatal mode).

Flow-triggering is selected by moving the trigger sensitivity level below PEEP into the green or red area. The baseline flow is 2.0 L/min (adult), 1.0 L/min (pediatric), and 0.5 L/min (neonatal). The flow-sensitivity setting is adjustable via an uncalibrated control. When the sensitivity is exceeded, flows of 180 L/min (adult), 30 L/min (pediatric), and 12 L/min (neonatal) maintain the baseline pressure (pressure limit). Inspiration is flow-cycled when inspiratory flow to maintain the pressure limit decays below 5% of initial peak flow or 80% of the set Breath Cycle Time.

Flow-triggering should not be used if leaks in the system are greater than the base flow because this causes auto-triggering.

Discussion

Sassoon has previously provided, in this Journal,[8] a wonderfully detailed discussion of the trigger variable. Much of the following discussion is based on her work.

Sassoon divides triggering into the trigger and post-trigger phase. Within the trigger phase, she lists a variety of factors that influence the responsiveness of the system, including errors due to the speed of the pressure signal, digital sampling of the pressure transducer, accuracy of the pressure transducer, discrepancies between set-PEEP and actual-PEEP, and noise in the patient circuit. Of these, the discrepancy between set-PEEP and actual-PEEP deserves clarification.

Most ventilators pressure-trigger based on the set baseline pressure—that is if PEEP is set at 5 cm H_2O and sensitivity is −1 cm H_2O, a breath is triggered when airway pressure falls below 4 cm H_2O. If leaks in the patient circuit or errors in PEEP control occur, auto-triggering or increases in patient work may occur (auto-triggering is often referred to

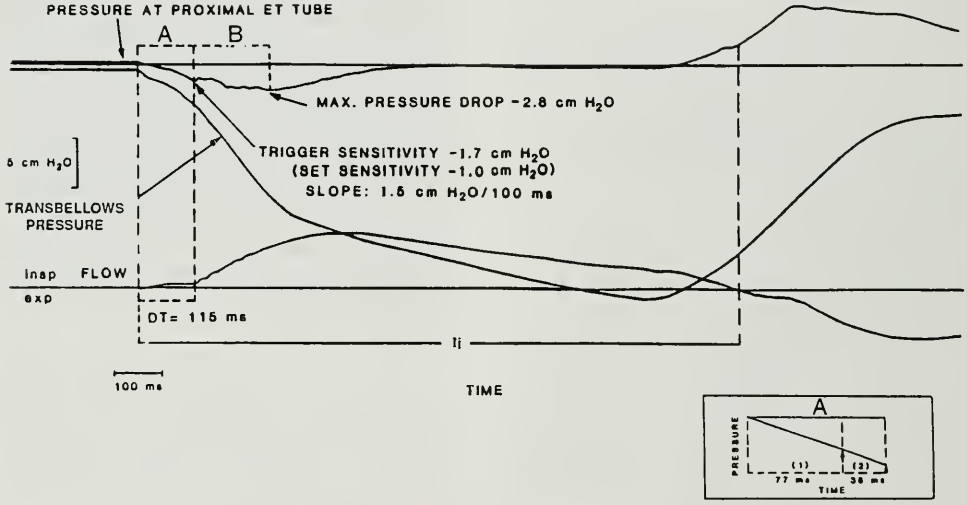

Fig. 2A. Pressure-triggered or demand-flow system applied to a mechanical lung model. Pressure is measured at the proximal end of the endotracheal tube (7.5 mm), and transbellows pressure and flow tracings are generated using a one-compartment computer-driven mechanical lung model (tidal volume of 500 mL, a rapid ascending and gradual descending ramp flow waveform at a rate of 45 L/min, and compliance of 25 mL/cm H_2O) ventilated with a demand-flow CPAP system set at a CPAP level of 0 cm H_2O without a humidifier (Puritan-Bennett 7200a). Part A, trigger phase = from onset of inspiratory effort to onset of flow delivery. Flow is slightly detectable during Part A as a result of circuit length and compliance. Part B, post-trigger phase = from measured trigger sensitivity to maximum pressure drop. Inset: A-1 is from onset of inspiratory effort to set sensitivity or trigger threshold; A-2 is from threshold to onset of inspiratory flow. See text for further explanation. CPAP = continuous positive airway pressure; DT = time delay from onset of inspiratory effort (negative deflection of transbellows pressure) to onset of flow; t_I = inspiratory time. (Reprinted from Reference 8.)

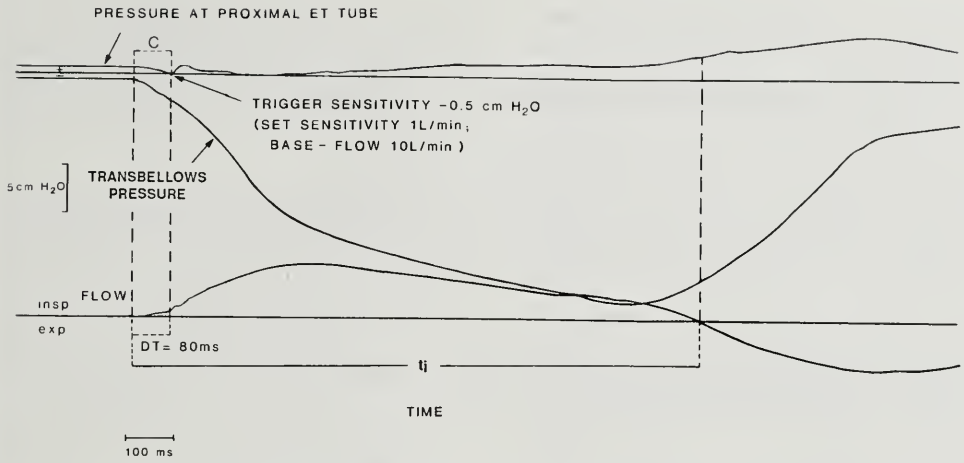

Fig. 2B. Flow-triggered or Flow-By system applied to a mechanical lung model at a CPAP level of 0 cm H_2O. Settings of mechanical lung model are the same as in Figure 2A. Part C = from inspiratory effort to opening of the pneumatic system. Flow during Part C is provided by the base flow. Note: Airway pressure remains at or above atmospheric pressure, acting as a small inspiratory pressure-assist due to the relatively optimal flow-control algorithm. The measured trigger sensitivity of 0.5 cm H_2O is not the actual sensitivity that triggers the pneumatic system to open. (Reprinted from Reference 8.)

as auto-cycling but because cycling is automatic termination of inspiration, auto-triggering is the preferred term). As an example, under the previously stated conditions if measured PEEP is 4 cm H_2O the ventilator auto-triggers. If measured PEEP is 7 cm H_2O, then the patient must generate 3-cm-H_2O negative pressure to trigger the ventilator. It is preferable to have the ventilator trigger from the pressure measured by the pressure transducer (as opposed to the set value); however, this leads to problems with identifying the PEEP level (due to signal noise) and to a whole host of new problems associated with signal conditioning and processing.

Another case in which set-PEEP and actual-PEEP are unequal occurs when auto-PEEP is present. In this instance, if set-PEEP is 5 cm H_2O, auto-PEEP (due to intrinsic airway disease and air-trapping) is 8 cm H_2O, and sensitivity is set at −1 cm H_2O, then the patient must generate −4 cm H_2O to trigger the ventilator. In this example, the presence of auto-PEEP and the need to generate −4 cm H_2O is unrelated to ventilator function and cannot be overcome by any new triggering mechanism. The only way to facilitate triggering in this instance is to balance set-PEEP and auto-PEEP.[11]

The post-trigger phase described by Sassoon includes the limit and cycle variables as well as the flow and attack rate of the flow as it approaches the limit variable. Understanding these concepts is critical to understanding the advantages of flow-triggering.

Sassoon has shown that the WOB is less with flow-triggering compared to pressure-triggering using the 7200ae in a lung model, in normal volunteers, and in patients.[8,9,12,13] She found that in a lung model, flow-triggering resulted in a shorter delay time between the onset of inspiratory effort and the onset of inspiratory flow compared to pressure-triggering (75 vs 115 ms) (Figs. 2 & 3). However, this slight improvement is not adequate to explain the decreases in the WOB seen clinically.

During pressure-triggering with the 7200ae, the limit variable is the PEEP setting minus the sensitivity—that is, if PEEP is 5 cm H_2O and sensitivity is −2 cm H_2O, the 7200ae attempts to maintain pressure at 3 cm H_2O throughout inspiration. During flow-triggering the limit variable is PEEP plus 0.5 cm H_2O—that is, at a PEEP of 5 cm H_2O, the 7200ae attempts to maintain pressure at 5.5 cm H_2O throughout inspiration. The difference is that during

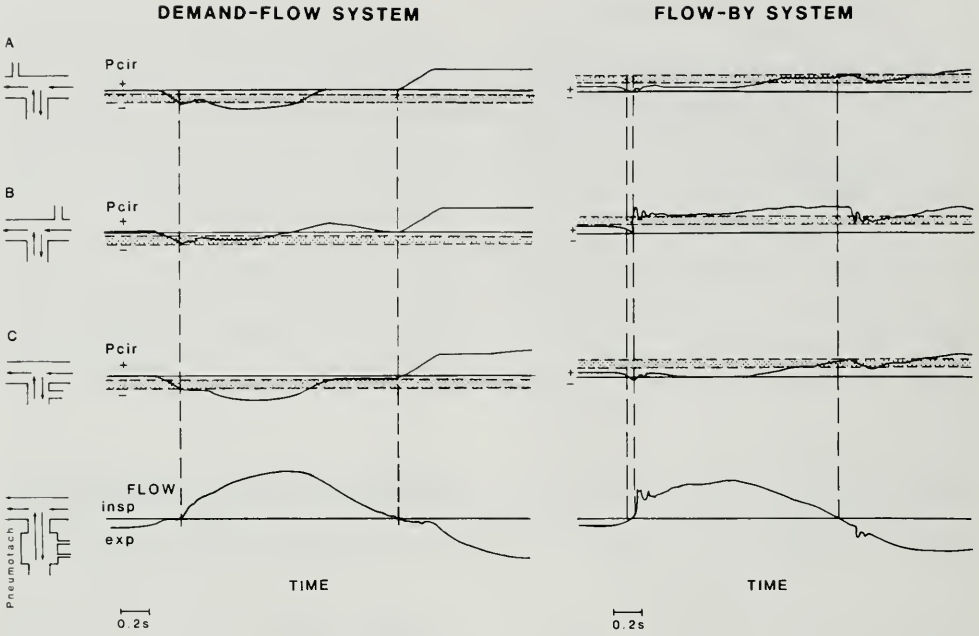

Fig. 3. Simulates measurements of pressures within the circuit using three pressure transducers placed at the exhalation port (A), inhalation port (B), and patient Y (C) during demand-flow and Flow-By CPAP. These data were obtained by having a normal subject breathe quietly through a mouthpiece at a CPAP level of 0 cm H_2O. A pneumotachograph was placed at the patient Y for measurement of flow. Note that the pressure-sensing location remains at the exhalation port. The pressure gradient between circuit pressure (P_{cir}) and the target pressure band (shaded area) is small when measured at the inhalation port (B). This pressure gradient represents the feedback signal to activate the flow-pressure control algorithm if the pressure-sensing line were located in the inhalation port. Notice that P_{cir} may overshoot target pressure with both demand-flow and Flow-By because of the inherently imperfect controller. (Reprinted from Reference 8.)

Flow-By, the patient receives what amounts to a small amount of pressure support compared to pressure-triggering.

The cycle variable is also different. During pressure-triggering, the 7200ae cycles into expiration when airway pressure exceeds PEEP by 1.0 cm H_2O and cycles during flow-triggering when flow through the expiratory flow transducer exceeds flow through the inspiratory flow transducer by 2 L/min.

The attack rate and maximum flow (180 L/min) for the 7200ae remain the same for pressure- and flow-triggering.

Work by MacIntyre and Ho[14] and by my colleagues and me[15] has shown that changing the attack rate or slope during pressure support can improve patient-ventilator synchrony. Similar adjustments can be made during spontaneous breathing using both the BEAR 1000 and Siemens 300 ventilators. These Percent Rise Time or Pressure Slope settings control how quickly the limit variable is reached during inspiration. A word of caution on these modifications. With a lung model, the fastest rise-time always results in the lowest WOB; however, in a clinical situation, small endotracheal tubes, elevated airway resistance, and diminished patient respiratory drive may dictate the use of slower rise-times. In these cases, the fastest rise-time may cause pressure overshoot and premature cycling of inspiration while the patient continues to inspire.

Laboratory comparisons of flow- and pressure-triggering systems are few. Hirsch et al found that with a CPAP of 0 and an inspiratory flow demand of

40 L/min, the WOB_I is 0.032 J/L for the 7200a in pressure-triggering and 0.022 for the 7200a in flow-triggering.[2] Although this represents a reduction in WOB_I of one third, the values are minute with respect to the normal work of breathing (0.5 to 0.7 J/L).

In a study of 9 patients, Sassoon found that flow-triggering with the 7200ae resulted in a significant decrease in the WOB (0.7 vs 1.0 J/L) compared to pressure-triggering with the same ventilator.[13] She attributed these changes to the beneficial effects of the baseline flow and to the fact that "flow is aggressively delivered during early inhalation."

We performed a similar study in a group of 10 patients. In our study, all patients were allowed to breathe on CPAP using the 7200ae and Veolar with both pressure- and flow-triggering.[16] Like Sassoon et al, we found that flow-triggering with the 7200ae resulted in a 25-30% reduction in the WOB compared to pressure-triggering. Analysis of the ventilator waveforms led us to a similar conclusion. During flow-triggering, the 7200ae triggers more quickly, attacks the target faster, and provides a slight amount of positive pressure during inspiration.

We also found reductions in the WOB with the Veolar of 30-40%, but from what we believe are different mechanisms. During pressure-triggering, the Veolar senses inspiratory effort on the inspired limb of the ventilator. This means the patient has to overcome the resistance of the breathing circuit and humidifier to trigger inspiration. With flow-triggering, measurements are made at the proximal airway flow transducer. This eliminates the humidifier from the equation and speeds the signal response time. As we have discussed, the Veolar uses identical limit and cycle variables for pressure- and flow-triggering. This is unlike the 7200ae, but flow-triggering with the Veolar is superior to its pressure-triggering mechanism.

In a recent abstract (1993), we compared the four adult ventilators that provide both flow- and pressure-triggering.[17] Using a dual-chambered lung model to simulate spontaneous breathing, we measured the WOB_I of the four systems discussed here at combinations of 0 and 10 cm H_2O CPAP and 0 and 10 cm H_2O PSV. We found that the flow-triggering systems for all ventilators except the Siemens 300 were superior to the pressure-triggering systems. The Siemens 300 had only a slight improvement with flow-triggering. Our results showed that the Siemens 300 had the lowest WOB_I followed by the 7200ae, Veolar, and 8400STi. Not surprisingly, the ventilator that maintained set baseline pressure, instead of shooting for a limit variable above baseline, had the highest WOB_I. However, the values we recorded for the lowest WOB_I and highest WOB_I for a given circumstance were small. In one instance the difference was 10-fold, 0.01 vs 0.001 J/L. This would appear to be a significant improvement; however, this represents a change in the total WOB (assuming normal WOB is 0.5 J/L) of only 1.8%. Whether this represents a clinically important change is a matter open to discussion and further study.

Flow-triggering appears to offer several advantages to pressure-triggering. The reasons for these differences depend on the nuances of the individual ventilators. Flow-triggering does appear to improve patient-ventilator synchrony and has been shown[17] in the 7200ae and Veolar to reduce the WOB clinically. Flow-triggering with the Siemens 300 and Bird 8400STi remains to be studied clinically.

Flow-triggering cannot reduce the WOB due to the endotracheal tube or auto-PEEP. The use of flow-triggering appears to be becoming a de facto standard. Respiratory care practitioners should understand the trigger, limit, and cycle variables associated with the ventilators they use and how they change when options are selected. The sophistication of microprocessor ventilators allows manufacturers to tinker with breath delivery in an attempt to provide the lowest WOB. I believe that the abilities of the ventilators described here are similar enough that the important fact is not what the ventilator does but that the respiratory care practitioner knows the ventilator's limitations and uses the ventilator correctly.

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Diseases of the Heart

The heart, from the important part which it plays in the animal economy, is subject to various, serious, and often fatal, diseases. Like the other viscera, it is removed from the eye, so that but little knowledge of its condition can be obtained by inspection; and hence we must have recourse to other means. The ear is the principal means of obtaining a knowledge of the state of the heart, and by auscultation and percussion, we are enabled to detect the existence of various diseases. The heart gives out two sounds, known as the first and second, which are distinguished from each other. The first sound is longer than the second, and the interval between the first and second sounds is shorter than that between the second and first. They have been compared to the two syllables 'lupp dupp.' Any manifest alteration in these sounds is indicative of the existence of disease. They may be high or low, clear or dull, muffled, rough, intermittent, etc. Murmurs or regurgitant sounds may arise from diseases of the valves. The power of distinguishing between the normal and abnormal sounds of the heart, and of the causes producing the latter, can only be obtained by lengthy experience. Diseases of the heart are usually divided into two classes: first, functional or nervous; and second, structural or organic. Chief among the former are palpitations, syncope or fainting, and angina pectoris. They are chiefly to be met with in persons of a naturally nervous temperament, more especially women suffering from hysteria or other like complaints, and may be induced by great mental excitement. In such cases, great attention should be paid to the general health, and, by means of tonics, sea-bathing, and gentle, open-air exercise, the system is to be strengthened. Violent exertion and strong mental excitement are particularly to be avoided. Among the principle organic diseases to which the heart is subject, are pericarditis, carditis, endocarditis, atrophy, hypertrophy, dilation, and valvular disease.

Heart Disease — Symptoms

Of all the diseases of the heart, the general symptoms are nearly the same. Respiration habitually short and constrained; palpitations and stiflings invariably produced by the motion of ascent, by rapid

This material is adapted from "Dr Chase's Third, Last and Complete Receipt Book and Household Physician," by AW Chase MD, which was published in 1903 by FB Dickerson Company of Detroit, Michigan, 1903:20,85,108,244-245.

These excerpts were made available to RESPIRATORY CARE by Crystal L Dunlevy EdD RRT, Assistant Professor and Director of Clinical Education, Respiratory Therapy Division, School of Allied Medical Professions, The Ohio State University, Columbus, Ohio.

walking, and by mental emotions, and returning without known cause; frightful dreams and interruptions of the sleep by sudden startings; and, lastly, a cachectic paleness, with tendency to leukophlegmatic effusion, which eventually appears, are all symptoms which, to a greater or lesser extent, occur in persons affected with diseases of the heart.

Treatment

In all cases of heart disease the body and mind should be kept as easy and cheerful as possible. The diet should be well-regulated—nourishing, but not stimulating. Coffee, tea, liquors, and tobacco must be dispensed with. The feet should be constantly warm and dry, and occasionally rubbed with mustard.

For inflammatory diseases of the heart, the bowels, if costive, may be moved with compound tincture of jalap. To each dose, add 10 grains of cream of tartar. Keep up a perspiration until the pain is relieved, by giving a teaspoonful of compound tincture of Virginia snake-root; also a warm infusion of pleurisy root. Mustard-plasters over the chest and spinal column are also to be employed. If the patient is troubled with sleeplessness, give 8 to 10 grains of compound powder of ipecac and opium.

For palpitation, the tincture of digitalis, 10 or 15 drops, 3 or 4 times a day, has been found useful. When the nervous system is affected, give small quantities of wine or spirits, or a few drops of laudanum or ether.

For neuralgia or breast-pang, give a teaspoonful of a mixture of equal parts of laudanum, ether, and oil of castor. The powder of Indian hemp-root may also be taken in doses of a small teaspoonful 2 or 3 times a day. If the stomach is acid, a teaspoonful of soda in half a tumbler of water will correct it.

Bear in mind that there are tinctures and compounds to cure all diseases, if we would but seek them out and test their virtues. Experiments on this subject too much neglected to afford us all of the information that we need.

Palpitation of the Heart — Cure For

As a simple cure for palpitation of the heart, hold the breath as long as possible and repeatedly. It is an almost certain remedy. In holding the breath, the blood is not invigorated by the absorption of oxygen in the air by its passage through the lungs, and hence, the blood does not pass freely nor quickly to the heart, and, therefore, its excessive action soon diminishes, and is finally quieted altogether.

Heart, Palpitation of, Fluttering, Etc. —Remedies

When persons become weak and feeble, from whatever cause, there is often a palpitation or fluttering of the heart, as many call it, from this weakness. In such cases, take any of our good tonics, as per directions, or obtain fluid extract of cereus bonplandi (a species of the cactus), 1/2 ounce. Take 10 drops at bedtime only, in a little water, and generally, relief will be soon realized, and a cure, permanent. I have so proved it. Continue to use the tonic remedies as long as needed.

Heart Disease, the Value of Buttermilk

In diseases of the heart, the French claim that buttermilk is invaluable; as the lactic acid in it dissolves and prevents ossification (bone-like condition) of the valves and arteries of the heart. It is worthy of a trial, and no doubt will prove valuable if continued faithfully for several months.

Bar Code Technology in Health Care: A Tool for Enhancing Quality, Productivity, and Cost Management, by Karen M Longe and Lisa Brenner. Softcover, illustrated, 130 pages. Cleveland OH: ADVANSTAR Communications, 1993. $29.95.

This book introduces the reader to information management that utilizes bar-code technology for its data entry vehicle. The book is written in a basic, simplistic way that allows a person with no previous exposure to bar codes to understand and walk away with an enhanced knowledge of the subject.

The Chapter that I read with the most interest was Chapter 2, which related the history of bar coding—the assumption, of course, being that the reader has an interest in bar-coding technology to begin with. A strength of the text for any application is Chapter 9, "Cost Justification: Dollars and Sense." This chapter gives interesting ideas on how to justify any technology required by the reader. "Making It Work: Preparing for Successful Health Care Applications," Chapter 10, provides insight into identifying the factors to be considered when preparing institutions for success in making any change.

The authors leave the impression (I believe wrongly) that bar coding is a solution to many healthcare operational issues. In Chapter 1, they refer to bar coding as an automatic data capture technology. Although bar coding is an automated process, it is far from automatic. Automatic suggests a laborless system, whereas automated can refer to the enhancement of a manual process. Limitations associated with any type of point-of-service data entry are minimized by the authors.

The definite weakness of the text is its failure to adequately emphasize the importance of the software associated with any type of information technology. The text continually dwells on hardware. Chapter 5 talks in detail of labeling, bar-code printers, and scanners, with only one paragraph dedicated to software. Chapter 7 talks extensively of hardware, radio frequency communication, voice recognition, pen-based computing, optics, and card technologies, although it also de-emphasizes the importance of software associated with any of this technology.

In summary, authors Longe and Brenner have put together an effective book that outlines considerations for obtaining information technology. However, at $29.95, the book may be a bit overpriced for the actual value of the information that can be obtained from it.

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Dailey's Notes on Blood, 2nd edition, by John F Dailey. Soft-cover, 178 pages, illustrated. Somerville MA: Medical Consulting Group, 1993. Bookshelf edition, $18.00; standard edition, $26.00.

The study of blood, its function and responsibility in human physiology, is for most of us a complex and extremely difficult undertaking, requiring a knowledge of both biology and chemistry. Because of this, standard texts on blood are themselves complex and difficult to understand. This comprehensive guide to understanding the basics of blood succeeds in being easy to use and comprehend, offering the student and clinician concise and timely information.

The book is divided into 18 chapters and discusses the origins, function, components, and disorders of blood. It also discusses blood transfusion, autologous blood recovery, component therapy, and volume expanders. Illustrations and figures accompany each chapter, and, although helpful, they are almost too simplified. Important terminology is highlighted in the margin of each page, allowing for quick location of material of interest. Each chapter contains study guide questions at its conclusion that are extremely helpful in identifying and reinforcing key points.

Chapters 1 and 2 introduce the concept and production of blood cells and the circulatory system, with a rather nice overview of the lymphatic system. Chapter 3 is concerned with explaining the immune system and its relationship to white blood cells. Antibody production, the five classes of antibodies, and both cell-mediated and humoral responses are well covered in this chapter, with white blood cells covered in-depth in Chapter 6. Chapter 5 offers a comprehensive review of erythrocytes, with Chapters 7-9 and 12 reviewing platelets, plasma, and their respective roles in hemostasis. Chapters 10 and 11 discuss, simply, the very complex subject of coagulation and coagulation disorders. Chapters 13-18 are devoted to blood replacement and volume expanders. The reader is deftly guided through individual discussions of blood transfusion, transfusion reactions, autologous blood recovery, and component therapy. Useful charts are included in Chapters 14 and 17.

Answers to the chapter questions and a thorough 19-page glossary are found at the end of the book. The appendix includes normal blood values, electrolyte values, clotting times, and a description of blood coagulation evaluation tests. What this book does not include is any discussion of fetal hemoglobin or circulation. Also,

hemodynamic values are not referenced to body surface area (BSA). Those respiratory care practitioners and nurses who work with pediatric patients would have found the added information helpful.

We highly recommend this book to respiratory care and nursing students and to medical residents, who need a comprehensive, yet easy, reference on the subject of blood. This is also an excellent resource for the respiratory care practitioner, nurse, and physician at the bedside, who often need quick access to clear and concise information in making clinical decisions—a welcome addition to any department's library.

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Pulmonary Diseases and Disorders, 2nd edition, edited by Alfred P Fishman MD. Hardcover, illustrated, 3 volumes, 2,563 pages. New York: McGraw-Hill, 1988. $300.00.

Pulmonary Diseases and Disorders, second edition, is the most recent and updated version of Fishman's encyclopedic text of lung diseases first published in 1980. The new edition sets out to update and to improve on that first 2-volume effort. The current edition has three volumes, and many subjects were added or the depth of coverage expanded. The 8-year hiatus between editions reflects the difficulties in updating textbooks that have an extensive coverage such as this one. There are more than 150 collaborators, all experts in their respective subjects, who must come together to provide the most recent information in their fields of expertise. The current edition though still up to date in the field of pulmonary physiology is, sadly, slightly behind the times in the areas of critical care and interstitial diseases. An attempt to counteract this was made by the publication of an updated volume, which was not available for my review.

The textbook is intended for clinicians interested in the field of pulmonary diseases. As Fishman's preface states, the aim is to offer a panoramic view of pulmonology without being too shallow in coverage. Respiratory therapists may find the chapters on physiology and pulmonary mechanics particularly informative.

The volumes are separated into 20 parts within which topics are interrelated in 167 chapters, each covering a subject with reasonable depth. The text is extensively illustrated, which helps with difficult concepts. The chapter on design and structure of the human lung has fantastic scanning electron micrographs of the lung, which aid in the visualization of the delicately balanced architecture of this organ. In general, the illustrations remain of high quality throughout the text, with the exception of a few 'fuzzy' photographs (usually radiographs, which are hard to reproduce). The organization of the text by chapter and section is logical and easy to follow. The index is well done without sending the reader on a 'safari' for the subject being searched.

Pulmonary Diseases and Disorders is a well-balanced treatise of pulmonology due to the number of experts who collaborated in this work. All pulmonary disorders and the associated mechanisms are carefully included and presented in a logical manner. In addition, disorders of the lungs are correlated to other organ systems of the body and integrated within the core of medicine as a whole.

Despite being such a complete work, the textbook does have its drawbacks, and these are evident in the outdated information on some disorders. One must remember that the text was published in 1988, with the most recent references prior to that dated 1987. The areas that suffer the most are the chapters dealing with the Acquired Immunodeficiency Syndrome, interstitial diseases of the lung, critical care (Special Therapeutic Interventions), and lung transplantation.

I believe that the textbook is a good general reference on pulmonary diseases and would be a good acquisition for medical libraries.The text is a sound base and reference for lung disorders, but may be too exhaustive for some readers. The text should be read to grasp understanding of the mechanism of pulmonary disorders but should be supplemented with more up-to-date information if the reader seeks state-of-the-art knowledge.

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Bronchial Asthma: Mechanisms and Therapeutics, 3rd edition, edited by Earle B Weiss MD and Myron Stein MD. Cloth bound, 1,259 pages, illustrated. Boston: Little, Brown & Co, 1993. $210.00.

It was Dr William Osler who adjured his students to "read the textbooks." His own textbook of medicine with its multiple revisions became standard reading fare for generations of medical students. Weiss and Stein have edited their textbook

of asthma for the third time within the last two decades and produced a massive tome that will serve well in the reference libraries of those interested in any aspect of asthma, from mechanisms of action, to diagnostics, to therapy and management.

In the section on mechanisms, significant emphasis is placed upon the exploding generation of information generated by basic scientists concerned with cellular, immunologic, and biochemical mechanisms of asthma, with research presented current to 1991. These chapters have little current practical clinical value, but they serve the purpose of presenting state-of-the-art minireviews of the inflammatory process in the airways of asthmatics, focusing on specific cellular components and mediators in addition to basic pathophysiologic and epidemiologic considerations.

The section entitled "Diagnostics and Laboratory" reviews clinical testing and the differential diagnosis of patients with asthma-like symptoms. Technical information and details provided are most helpful. For example, Spector's Chapter 39 on bronchoprovocation includes a brief discussion of the diluent reaction in asthmatics undergoing methacholine challenge, which is barely mentioned in some references on challenge testing. Brooks' review of occupational asthma is not only comprehensive but the appended references are extensive and relevant. Significant in its omission as a chapter in itself is the discussion of vocal cord dysfunction mimicking asthma, a subject upon which much interest is currently centered. Likewise, the role and mechanism of metabisulfite-triggered asthma is not sufficiently covered and needs to be more extensively discussed and referenced in a future edition.

The chapters in the section "Therapy and Patient Management" are certainly complete, although some topics have little current practical application (eg, Chapter 71, Calcium, Calcium Antagonists, and Oxyradicals), and some contributors disagree among themselves concerning the relevance and efficacy of different therapies. It is not surprising that Mullarkey writes of the potential role of methotrexate in steroid-dependent asthma (Chapter 68); Zeiger and Szefler review the usefulness of TAO/Medrol (Chapter 67); but Ziment and Stein include both drugs within their chapter on "Inappropriate and Unusual Remedies" (Chapter 88).

A major omission, which may be related to the timing of publication, is the authors' failure to mention the National Asthma Education Panel Report (NAEP) published by the NHLBI in the Fall of 1991. This report currently represents the practice standard for the diagnosis and treatment of asthma. Failure to discuss the NHLBI Guidelines diminishes the relevancy of this text. Faling and Snider's grading of asthma severity (Chapter 54) uses a set of criteria quite different from the NAEP, which would alter the recommended medical management of many asthmatics, and may be confusing in years to come when severity scales are used to guide treatment choices. NAEP's use of inhaled anti-inflammatory agents early in the management of asthma, sharply differs from Faling and Snider's lukewarm feelings toward use of this class of agents in many asthmatics. Fortunately, Toogood et al's discussion of inhaled anti-inflammatory agents (Chapter 61) more than compensates for the "old school" conservatism of Faling and Snider and cites relevant data from the medical literature and their personal experiences to emphasize the pre-eminent role that inhaled anti-inflammatory agents now play in the management of asthma. It would have been helpful if Toogood et al had cited more literature and experience with inhaled medications other than beclomethasone and budesonide.

Its sheer size makes reading Weiss and Stein from cover to cover an impossible task, and the desire to present as complete a textbook as possible brings with it inherent problems of topic organization and relevancy to the general reader. In future editions, some chapters should be consolidated. Selective readings of chapter groupings would help familiarize a reader with the breadth of information presented. This work should be a valuable reference for all students of respiratory disease: respiratory care practitioners, nurses, and physicians.

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Monitoring in Respiratory Care, edited by Robert M Kacmarek PhD RRT, Dean Hess MEd RRT, and James K Stoller MD, with 38 contributors. Hardcover, 841 pages, illustrated. St Louis: Mosby-Year Book Inc, 1993. $52.95.

When I first glanced at the cover of this book, I was taken by two items of interest. First, the title told me that this was a different type of book—a unique book for respiratory care. **Monitoring in Respiratory Care** does not deal with the general topics that have been so well attended to in the publications of recent years; rather the editors have found a previously unfilled niche in the respiratory care market. The second detail that attracted my attention was the names of the editors. In addition to having been listed on the Editorial Board of RESPIRATORY CARE journal, these are names that are prominently and perpetually displayed on the Medline 'marquee.' Not only are the editors some of the most well-respected au-

thors and educators in the field of respiratory care, but they also have compiled a cast of contributors familiar to the avid reader of respiratory and anesthesia literature.

The editors (Kacmarek, an Assistant Professor in the Department of Anesthesia at Harvard Medical School, and Director of Respiratory Care at Massachusetts General Hospital; Hess, an Instructor in Harvard's Department of Anesthesia and Assistant Director at Massachusetts General; and Stoller, the Medical Director and Head of the Section of Respiratory Care in the Department of Pulmonary and Critical Care Medicine at the Cleveland Clinic Foundation) are familiar with the wealth of technologic data that has inundated the field of respiratory care, having themselves been responsible for a respectable portion of this information. This text's 26 chapters provide a comprehensive review of cardiopulmonary monitoring for the respiratory practitioner that, to this reviewer's knowledge, has not previously been collected under one title.

The book is divided into four sections beginning with Overview and Engineering Principles (Section 1). This section offers the reader a background for the concepts and principles inherent to monitoring. The individual chapters are Perspectives on Monitoring in Respiratory Care, Fluid Mechanics, Sensors and Transducers, Signal Processing and Data Display, Fundamentals of Metrology: Evaluation of Instrument Error and Method Agreement, and Quality Assessment and Maintenance of Respiratory Monitors.

Section 2, Invasive Monitoring Techniques, includes Oxygen Transport from Lung to Tissue, Hemodynamic Monitoring, and Work of Breathing and Respiratory Muscle Function. The chapters devoted to noninvasive techniques (Section 3) are Pulse Oximetry; Transcutaneous Oxygen and Carbon Dioxide Measurements; Capnography: Technical Aspects and Clinical Applications; Indirect Calorimetry; Cardiovascular Function; Impedance Pneumography, Apnea Monitoring, and Respiratory Inductive Plethysmography; Airway Pressure, Flow and Volume Waveforms, and Lung Mechanics during Mechanical Ventilation; Measurement of Lung Volume at the Bedside; and, The Computer for Charting and Monitoring.

Section 4, Integration of Monitoring into Various Clinical Settings, incorporates the ideas introduced in the earlier sections into clinical practice: Respiratory Monitoring in the Neonatal Intensive Care Unit, Monitoring in Pediatric Care, Monitoring in Adult Critical Care, Monitoring in Adult General Care, Monitoring in Anesthesia, Monitoring in the Home and the Outpatient Setting, Unattended Monitoring for Respiratory Events, and Monitoring in High-Altitude Environments.

I formed a number of opinions of this book that varied according to the individual chapters. The editors, in view of the knowledgeable authors, justifiably allowed divergent styles and formats for each chapter. Certain chapters were particularly informative and appealing. Chapter 3 (Sensors and Transducers) is concise, focused, and thorough in introducing the various mechanisms by which we monitor the patient. Numerous illustrations and charts nicely complement the written text.

I found myself questioning the appropriateness of including Chapter 5 (Fundamentals of Metrology: Evaluation of Instrument Error and Method Agreement) in a book devoted to monitoring—feeling that its proper appointment belonged in a medical statistics text; however, after reading this chapter, the role of measurement theory in monitoring practice to guide the proper evaluation and utilization of equipment became apparent.

Chapter 8 (Hemodynamic Monitoring) offers detail in a manner that I appreciate; indications, 'how-to,' and complications are presented in a fashion that can be quickly assimilated. The author effectively utilizes tables and illustrations to offer step-by-step directions relating to technique.

For the practitioner with limited pediatric exposure, Chapter 20 (Monitoring in Pediatric Care) offers a nice review in an easy-to-read format. The authors begin with pediatric anatomy and physiology and the physical examination before covering such topics as pulmonary function, indirect calorimetry, and the requisite review of cardiopulmonary monitoring techniques.

Chapter 26 (Monitoring in High-Altitude Environments) is an interesting chapter on hypobaric conditions. However, I can't help but wonder why this chapter wasn't expanded to include monitoring in hyperbaric environments because respiratory therapists are participants in patient care under this condition.

Not all chapters were as reader-friendly as those already mentioned. Much of Chapter 7 (Oxygen Transport from Lung to Tissue), especially the section concerning ventilation-perfusion mismatching, was indecipherable to me. I felt this chapter to be unnecessarily difficult to read and understand. Several aspects bothered me: (1) poor progression between text and tables—I found at least 7 errors here, principally missing data and reference to the wrong table; (2) errors in referencing—references were apparently numbered incorrectly; (3) chaotic and inconsistent use of abbreviations; (4) mistakes in the tables including reference, mathematical, and spelling errors; and (5) a number of spelling and/or typographical errors within the text.

Chapter 21 (Monitoring in Adult Critical Care) offers good data, yet is somewhat more challenging to read than most of the other chapters in part because of errors and inconsistencies: incorrect citation of tables and equations, failure to identify abbreviations

at first use, and inconsistencies in abbreviations.

A plus for this book is the strong referencing to support the data offered in text. However, the reference lists at the end of each chapter have numerous errors and inconsistencies. There are too many to list but they include misspellings, inconsistent referencing style (varying between and within chapters), and citation of incorrect page numbers.

In summary, this book has both strengths and weaknesses. The numerous technical or mechanical flaws give the appearance of hurried production. The reference lists (and also the tables and figures) did not receive the same level of scrutiny that the text received. This makes it difficult for a reader who wishes to retrieve references (as I attempted) for further study. To paraphrase a statement relating to manufacturers (from Page 95 of this book), as authors and editors who are involved with medical publications, we have a responsibility to develop texts that are user-friendly, cost-effective, reliable, and, above all, accurate.

The book's content relating to monitoring in respiratory care is thorough and informative. It will appeal to the practicing respiratory therapist, intensive care nurse, physician intensivist, and respiratory therapy student. Overall, this is a good book with a wealth of information—one that, to this reviewer will be, and already has been, a frequently referenced book on the subject of patient monitoring.

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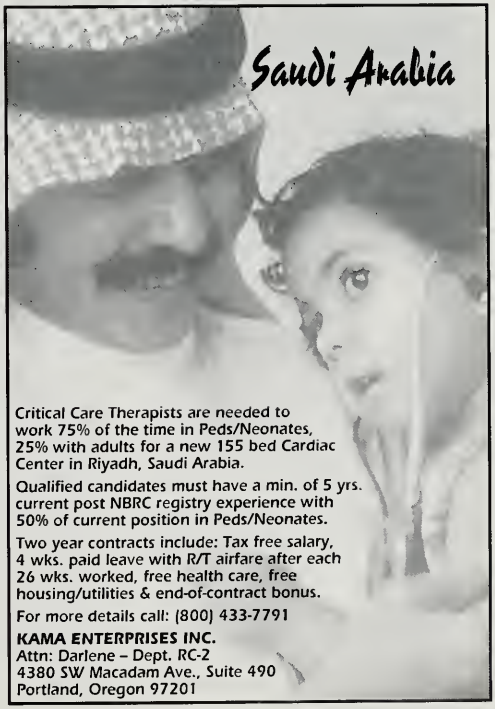
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Commentary on RCP Smoking Behavior

Congratulations on the excellent editorial "Walking the Walk—Not Just Talking the Talk: Cigarette Smoking and Respiratory Care Practitioners,"[1] which appeared in the October 1993 issue of RESPIRATORY CARE. As an RCP with over 16 years in the field, I wholeheartedly agree. In fact, I completed my Master's thesis on this topic.[2]

I had always been perplexed as to why so many of my peers continued to smoke after witnessing its sequelae firsthand on a daily basis during their respiratory care training. As a graduate student in Public Health Education at the University of North Carolina-Greensboro, I was obliged to review what seemed like all of the literature hypothesizing why humans do or do not engage in preventive health behaviors. Several authors addressed the inevitable problems that face health educators confronted with target groups who lack salient images of the documented consequences of smoking.[3,4] But, why don't these salient images seem to have a greater impact on smoking by respiratory care practitioners (RCPs)?

I also learned about other factors possibly influencing health-related decision making. Another intriguing (and seemingly related) theory was that of the so-called availability heuristic.[5] In a nutshell, it states that we routinely engage in a cognitive procedure for estimating frequency or probability based on the ease with which instances from a group or category are recalled. Certainly RCPs can recall instances of smoking-related morbidity and mortality without much strain, yet so many of them continue to smoke. Why?

These burning questions (and the need to complete a scientific study for my thesis) eventually led me to survey 294 registered therapists (RRTs) residing in North Carolina during July 1986 (which I estimated to be at least 80% of those practicing in the state at that time). The only literature I located that specifically documented the smoking behavior of RCPs was "Survey on RT Lifestyles," which appeared in the June 1986 issue of *AARC Times*.[6] That article reported that 17% of the 275 therapists and technicians comprising the sample group (derived from the AARC membership roster) were current smokers. Charting what seemed to be previously untouched territories, I set out not only to describe the prevalence of cigarette smoking among RRTs in North Carolina but also to test three hypotheses:

(1) RRTs overestimate smoking-related morbidity because of the action of the availability heuristic,

(2) RRTs who are exsmokers have higher empathy scores than do currently smoking RRTs (based on Hogan's Empathy Scale),[7] and

(3) RRTs who smoke are not influenced by the availability heuristic to quit because of a lack of empathy for the patients with whom they work.

A surprising 215 questionnaires (73.1%) were returned, thanks to endorsement by the North Carolina Society for Respiratory Care. Because my sample group consisted only of RRTs in a state where tobacco is the Number 1 cash crop, the generalizability of my findings to other populations may be limited. Some of the more interesting results included

- 25% of the sample group were found to be current smokers (compared to 27.4% of the general population of North Carolina at that time), and 21% were exsmokers.[8]
- of current and exsmokers, 8.2% started smoking during their RC training; another 6.2% started after graduation from an RC program.
- 10% of the respondents stated that their medical director smoked, and approximately 73% reported that at least one of their RC instructors smoked.
- 79% of respondents felt that it is an RCP's professional responsibility to set an example for patients and the public by not smoking (falling to 54% when examining only current smokers).
- 44.7% of respondents felt that their RC training did not include sufficient depth in smoking and its long-term consequences.
- Hypothesis 1 received only partial support (RRTs did significantly overestimate a smoker's relative risk for developing coronary heart disease; but, alas, it seemed that most respondents didn't understand the concept of relative risk).
- Hypothesis 2 was unsupported; in fact, I found the mean empathy score of all respondents to be lower than those previously published for other healthcare workers!
- Hypothesis 3 was also unsupported statistically (only 1.9% of the current smokers reported "never picturing themselves in the same position as patients they treat" versus 34.2% of the never-smokers).

So, not surprisingly, my thesis findings didn't change the world. I also was dismayed—yet not surprised—at documenting such a dismal percentage of smoking RC instructors. I do believe, however, that even 7 years later most of what I

wrote in the "Implications" section of my thesis remains true.[2]

> Perhaps the most important point that has been re-emphasized by this study's findings is that health knowledge has limited effects on health behaviors. It was first thought that if people only knew about the health consequences of smoking they wouldn't smoke. Next it seemed that many smokers required a more salient image of the health consequences of smoking in order to sufficiently motivate them to quit. It is clear from this study that for at least one fourth of the population this isn't enough. Consistent with most smokers surveyed throughout the literature, nearly all currently smoking RRTs reported wanting to quit. It may be that the availability heuristic has sufficiently motivated them to do so, but barriers to cessation might be more intense among this population than the general public (eg, occupational stress from working odd hours, performing complex tasks in pressure situations, caring for dying patients, social networks consisting of other knowledgeable smokers, etc.) . . .. It is certain that merely the dissemination of health information and even the addition of salient images of smoking sequelae is not sufficient to deter some individuals from smoking.

It would seem then that future anti-smoking efforts need be directed more strongly at prevention—especially before the high school years—and at improving cessation programs.

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More on Cigarette Smoking and RCPs

Your editorial "Walking the Walk—Not Just Talking the Talk: Cigarette Smoking and Respiratory Care Practitioners"[1] was both poignant and needed.

At a recent seminar seeing my state association president "out-back-for-a-smoke" caused me to react negatively and made me wish I had not helped to elect her.

Because voters often are not personally acquainted with the candidates, it would be helpful if the ballot included information as to whether the candidate is or is not a smoker.

Dixie Suzor CPFT RRT
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A "Statistically Significant" Response

A letter recently published in RESPIRATORY CARE[1] suggests that cold viruses may enter the nasal mucosa because of inadequate humidification. In his letter, Groff described how daily use of a saline nasal spray decreased his incidence of colds from 2 colds/year to only 2 colds during the 5-year period in which he used the nasal spray. He clearly indicated that he himself constituted the single subject (n = 1) in an informal design. He further said that his results "may not be statistically significant" but hoped that someone in the scientific community would further investigate this phenomenon.

I am especially interested in Groff's use of the term "statistically significant" because he did not report that he had performed any statistical tests on his data. Interestingly, his study may have revealed statistically significant results if one is willing to make a few plausible assumptions. First, let us take as the fundamental assumption that Groff had 2 colds in each of the 5 years prior to the nasal-spray (intervention) period, for as he states: ". . . I have always contracted 2 colds a year, one in late Fall and another in early Spring." That amounts to a preintervention series of 5 observations, each with 2 events of interest (2 colds/year in each of the last 5 years). Let us further assume a post-intervention series of another 5 observations with a total of 2 events or a mean of 0.4 colds/year.

Is there a statistical analysis that is appropriate for these data? The answer is—possibly. Because of (1) the likely dependence within and between the series and (2) the lack of normality because of the small number of events within each series, a parametric analysis probably is not valid.[2,3] For example, a 1-sample test of the hypothesis that the postintervention mean is equal to 2.0 colds/year (the preintervention mean)

yields a t statistic that *is* statistically significant ($p < 0.05$). This result holds whether the 2 postintervention colds are assumed to have occurred during the same year or in different years. If we drop the normality assumption but retain the questionable independence assumption for these data, one finds that both an exact nonparametric permutation test and the binomial sign test confirm the preceding statistically significant result. Finally, let us dispense with both of the preceding assumptions and perform a time-series analysis that takes into account the autocorrelated nature of the data. Although a larger number of events would have been desirable for both the pre- and postintervention series, the analysis was performed on the 10 observations reported (n = 5 postintervention) and implied (n = 5 preintervention) by Groff. Using the condition that the 2 postintervention colds may have occurred in the same year, 1 out of 5 possible permutations of the postintervention data yields a statistically significant t statistic based on the difference in the means. Using the more likely condition that the two postintervention colds occurred in different years, 7 of the 10 t statistics are statistically significant ($p < 0.05$).

These examples show that valid statistical tests can be performed on data such as Groff's. As was shown here, formal analyses of the data as described may actually provide support for the author's conclusion. However, the results of single "n = 1" studies cannot be applied to anyone else, and additional, more carefully controlled research on the nasal-spray hypothesis is required to establish the credibility of the conclusion. That is, besides statistical significance, researchers must satisfy certain methodologic criteria in order to persuade others that their conclusions are sound—in this case, that the reduction in colds is truly a result of the use of the nasal spray. While Groff's work lacked the methodologic rigor of a controlled clinical trial, he used the term "statistically significant" incorrectly because, as we have just seen, his results may well be statistically significant.

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REFERENCES

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Partial- vs Nonrebreathing Masks

I am writing to point out an omission that was made by Branson in his article entitled "The Nuts and Bolts of Increasing Arterial Oxygenation: Devices and Techniques."[1] In that article, he clearly explains the deficiencies of partial- and nonbreathing masks but fails to cite a relevant paper published by my colleagues and me "Delivery of High Inspired Oxygen by Face Mask,"[2] which looked at the performance of these masks. We showed that with human subjects breathing over a broad range of tidal volumes and frequencies the partial-rebreathing mask outperformed the nonrebreathing mask. This was thought most likely to be the result of the resistance of the one-way valve in the rebreathing mask. For a comfortably fitted mask, this resistance is believed to be the cause of the greater entrainment of room air that results in a lower F_{IO_2}.[3]

We also demonstrated that if the oxygen flow is less than 25 L/min, the subject may empty the reservoir, necessitating a higher oxygen flow to maintain the same oxygen concentration.

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Commentary on RETROSPECTROSCOPE

In the December 1993 issue of RESPIRATORY CARE, I greatly enjoyed reading the reprint of Julis H Comroe's article "Inflation—1904 Model," which described the attempts of Sauerbruch and other pioneer thoracic surgeons to solve the "pneumothorax problem."

Anyone interested in reviewing the full range of devices used—from the negative pressure chambers through the early ventilators up to those similar to the modern pattern—may wish to consult Mushin and Rendell-Baker's monograph on the Origin of Thoracic Anesthesia recently reissued by the Wood Library-Museum of the American Society of Anesthesiology, 520 Northwest Highway, Park Ridge IL 60068-2573.

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Loma Linda University
Loma Linda, California

The American Respiratory Care Foundation Awards for 1994

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$2,000 for the best original paper (study, evaluation, or case report) accepted for publication from December 1993 through October 1994. This award is not limited to papers based on OPEN FORUM presentations.

Four awards of $1,000 each for papers accepted for publication from November 1993 through October 1994 based on any OPEN FORUM presentation (not limited to 1993 OPEN FORUM).

Five awards of $500 each for the best papers submitted (not necessarily published) by 1994 OPEN FORUM participants who have 'never published' in the Journal. The never-published first author must present the abstract at the Annual Meeting and must submit a paper based on the abstract before the 1994 Annual Meeting (received in the Editorial Office by November 1, 1994). Co-authors may have previously published in RESPIRATORY CARE.

Dr Allen DeVilbiss Literary Award

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All awards will be made at the 1994 Annual Meeting. Papers are judged automatically. No application is necessary.

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RPFT:	December 3	September 1
Perinatal/ Pediatric:	March 12	November 1

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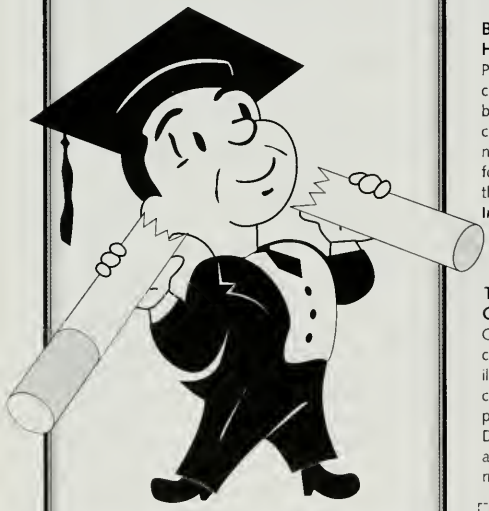

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Calendar of Events

AARC & AFFILIATES

February 22-25 in Reno, Nevada. The NSRC and the American Lung Association of Nevada present the 13th Annual High Sierra Critical Care Conference at the Peppermill Hotel Casino. The 4-day conference presents critical care topics covering adult, pediatric, and neonatal issues. Contact Colleen Banghart, American Lung Association of Nevada, PO Box 7056, Reno NV 89510. (702) 829-5864 (9 am to 3 pm PST).

March 29 AARC Videoconference. The AARC, in conjunction with VHA Satellite Network, presents the second of a 6-part videoconference series titled "Professor's Rounds in Respiratory Care." The second presentation is entitled "Therapist-Driven Protocols: Implementation" (One CRCE Credit). For information, call (214) 830-0061.

April 11-13 in Philadelphia, Pennsylvania. The PSRC presents its 29th annual conference and exhibition at the Adam's Mark Hotel. This year's theme is "Winds of Change." State Sputum Bowl finals are featured. Contact Betsy Schneck (215) 829-3578.

May 3-6 in Grand Rapids, Michigan. The MSRC holds its 1994 Scientific Symposium at the Amway Grand Plaza Hotel. Contact MSRC, PO Box 950, East Lansing MI 48826-0950, or call Caroline Kimmel (517) 336-7605.

May 11 AARC Videoconference. The AARC, in conjunction with VHA Satellite Network, presents the third of a 6-part videoconference series titled "Professor's Rounds in Respiratory Care." The third presentation is entitled "New Strategies for Asthma Management" (One CRCE Credit). For information call (214) 830-0061.

June 8-10 in Checotah, Oklahoma. The Oklahoma Society for Respiratory Care presents its 29th Annual State Seminar and Exhibition at Fountainhead Resort and Convention Center on Lake Eufaula. This year's theme, "The Times–They Are A Changin," addresses anticipated changes in the healthcare delivery system and critical issues for respiratory care. Other activities include a Manager's Mixer and the traditional golf tournament. For information on the seminar or exhibits contact Karen Hart RRT at (405) 273-6442.

July 26 AARC Videoconference. The AARC, in conjunction with VHA Satellite Network, presents the fourth of a 6-part videoconference series titled "Professor's Rounds in Respiratory Care." The fourth presentation is entitled "Arterial Blood Gas Trouble Shooting: Interpretation, Analysis, and Quality Control" (One CRCE Credit). For information call (214) 830-0061.

OTHER MEETINGS

March 5-8 in Glasgow, Scotland. The International Congress on Smoking Cessation is held. Contact Jane Fensome, Congress Secretariat, Gardiner-Caldwell Communications Ltd, The Old Ribbon Mill, Pitt Street, Macclesfield, Cheshire SK11 7PT, UK. Tel: 44 625 615325. Fax: 44 625 616563.

March 15-16 in Durham, North Carolina. Eighth Annual Cardiopulmonary Technology Seminar is sponsored by Duke University Medical Center, Durham Regional Hospital, Durham Technical Community College, and University of North Carolina Hospitals Respiratory Care Departments. Featured speakers are Michael Newhouse MD on Aerosol Delivery Systems for Pulmonary Medications, Neil MacIntyre MD on Intrapulmonary Pressure Graphic Analysis and Weaning, James Donohue MD on Advances in Respiratory Pharmacology, and Kathryn Ellis MSN on Manpower Changes in an Era of Health Care Reform. The seminar also features a comprehensive exhibit hall, where the latest advances in medical scientific equipment and services are displayed. Registration for both days is $70.00 including lunch on day one. Contact Susan Rinaldo-Gallo RRT at (919) 681-2720 or Chuck Alford at (919) 470-5366.

March 17-19 in Destin, Florida. Tulane School of Medicine in New Orleans, Lousiana, The University of Alabama at Birmingham, and the University of Florida presents "Practical Issues in Pediatric Home Care: An Interdisciplinary Approach" at the Sandestin Beach Hilton, Destin FL. The purpose of this conference is to provide information and resources to healthcare professionals working in the hospital and community setting with chronically ill and technology-dependent children. The care of children with pulmonary disease is discussed specifically from an interdisciplinary perspective. Approved for 13 hours of continuing education credit by the AARC. Fee: $125/$75 for employees of Title V Affiliated Agencies. Contact Pamela Rogers MSW or Anna Chiappetta RRT RCP, Pediatric Pulmonary Center, Tulane School of Medicine, Box SL 37, 1430 Tulane Ave, New Orleans LA 70112. (504) 588-5601.

March 21-23 in Cleveland, Ohio. The Cleveland Clinic Foundation sponsors a continuing education program

"Frontiers in Pulmonary and Critical Care Medicine" in Bunts Auditorium. Call 800-762-8173 for more information.

March 24-26 in Helena, Montana. The American Lung Association of Montana presents its 13th Annual Big Sky Pulmonary Ski Conference. Contact The American Lung Association of Montana, 825 Helena Ave, Helena MT 59601. (406) 442-6556, fax (406) 442-2346.

April 15-17 Miami to Nassau. Sunset Seminars is happy to anounce another Weekend Getaway "Floating Seminar" on a 2-night cruise aboard the SS Britanis with George Burton MD discussing "Therapist-Driven Protocols." 6 CE credits are awarded. Contact Sunset Seminars/Robbins & Associates, 19800 SW 180 Ave #127, Miami FL 33187. Call (305) 232-1908.

April 21-22 in San Diego, California. Round-Up your rehab skills by attending the California Society for Pulmonary Rehabilitation's 5th Annual Educational Seminar at the beautiful U.S. Grand Hotel. Speakers discuss exercise and nutrition, osteoporosis, ethical issues, allergy and environmental control, depression in COPD, and much more. Also join us for a country/western reception and dance. Contact Mary Jo Goldzimer RN RCP, (619) 759-1513, or Ann Devine BS RRT (909) 884-6673.

May 10-11 in Little Rock, Arkansas. Respiratory Care Services of Arkansas Children's Hospital presents the 1994 Diamond Conference. Special sessions focus on neonatal/pediatric airway topics and pre-ECMO strategies in acute respiratory failure, including nitric oxide, HFOV, and conventional ventilation. The meeting also features a poster session, lunch, tutorials, and roundtable discussions. Contact Mike Anders RRT, Respiratory Care Services, Arkansas Children's Hospital, 800 Marshall St, Little Rock AR 72202. (501) 320-3535, fax (501) 320-3411.

August 31-September 3 in Christchurch, New Zealand. The Third Congress of the Asia Pacific Association for Respiratory Care presents "Breathing into 2001," at the Christchurch Town Hall, Christchurch, New Zealand. Contact Stephanie K Humphries, The Planit Group Limited, Event Organizers, 201 Cambridge Terrace, Christchurch, New Zealand. (643) 366-5955, fax (643) 366-5944.

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1994 Call for Abstracts

RESPIRATORY CARE • OPEN FORUM

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present papers at the OPEN FORUM during the AARC Annual Meeting in Las Vegas, Nevada, December 10-13, 1994. Accepted abstracts will be published in the November 1994 issue of RESPIRATORY CARE. Membership in the AARC is not necessary for participation.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report (1) an **original study,** (2) the **evaluation of a method or device,** or (3) a **case or case series.** Topics may be aspects of adult acute care, continuing care/ rehabilitation, perinatology/pediatrics, cardiopulmonary technology, health occupations education, or management of personnel and health-care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a paper at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

Essential Content Elements

An **original study** abstract *must* include (1) Introduction: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results. A **method/device evaluation** abstract *must* include (1) Introduction: identification of the method or device and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience *or* a notation of lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate. A **case report** abstract *must* report a case that is uncommon or of exceptional teaching/learning value and must include: (1) patient data case summary and (2) significance of case. Content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

Abstract Format and Typing Instructions

Accepted abstracts will be photographed. First line of abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location. Underline presenter's name. Type or electronically print the abstract *single spaced in the space provided on the abstract blank.* Insert only one letter space between sentences. Text submission on diskette is encouraged but must be accompanied by a hard copy. Identifiers will be masked (blinded) for review. Make the abstract all one paragraph. Data may be submitted in table form *and simple figures may be included provided they fit within the space allotted. No* figures, illustrations, or tables are to be attached to the abstract form. Provide all author information requested in right column of abstract form. A clear photocopy of the abstract form may be used. Standard abbreviations may be employed without explanation. A new or infrequently used abbreviation should be preceded by the spelled-out term the first time it is used. Any recurring phrase or expression may be abbreviated if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to the editorial staff of RESPIRATORY CARE at (214) 243-2272.

Deadlines

Deadline Allowing Revision

Authors may choose to submit abstracts early. Abstracts received by March 15 will be reviewed and the authors notified by April 22. Rejected abstracts will be accompanied by a written critique that should in many cases enable authors to revise their abstracts and resubmit them by the final deadline (May 28).

Final Deadline

The mandatory Final Deadline is May 28 (postmark). Authors will be notified of acceptance or rejection by *letter only*— to be mailed by August 15.

Mailing Instructions

Mail (Do not fax!) 2 clear copies of the completed abstract form and a stamped, self-addressed postcard (for notice of receipt) to:

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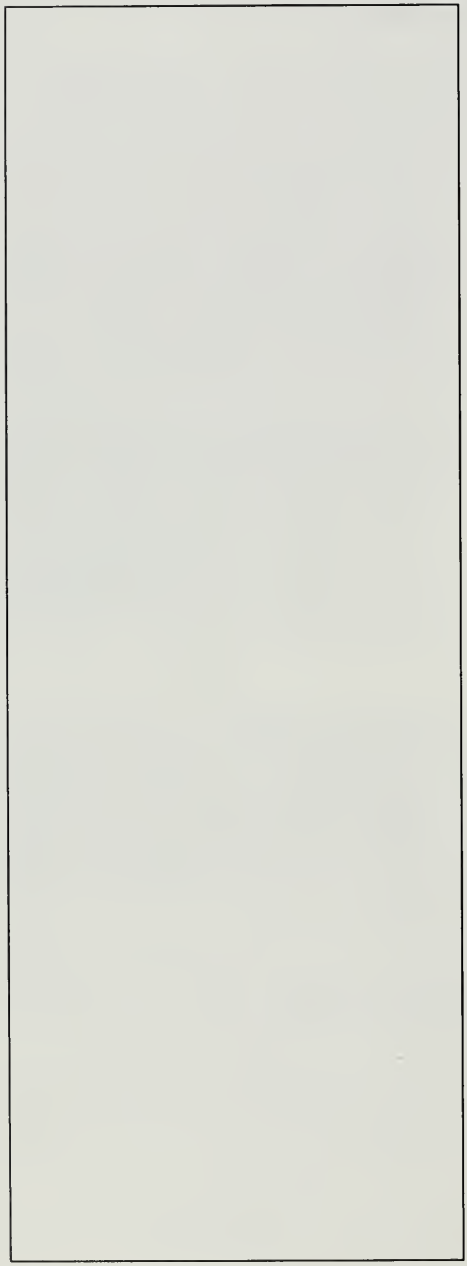
1994 Respiratory Care Open Forum

Abstract Form



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General Information

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Overview: A critical review of a pertinent topic about which not enough has been published to merit a Review Article.

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Editorial: A paper drawing attention to a pertinent concern; it may present an opposing opinion, clarify a position, or bring a problem into focus.

Letter: A signed communication about prior publications in this journal, or about other pertinent topics. Tables and illustrations may be included. Type double-spaced, supply a title, mark "For publication."

Blood Gas Corner: A brief, instructive case report involving respiratory care blood data—with questions, answers, discussion.

PFT Corner: Like Blood Gas Corner, but involving pulmonary function tests.

Test Your Radiologic Skill: Like Blood Gas Corner, but involving pulmonary medicine radiography and including one or more radiographs, may involve imaging techniques other than conventional chest radiography.

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Authorship: All persons listed as authors should have participated in the reported work and the shaping of the manuscript; all should have proofread the submitted manuscript; and all should be able to publicly discuss and defend the paper's content. A paper with collective (corporate) authorship must specify the key persons responsible for the article. Authorship is not justified solely on the basis of solicitation of funding, collection or analysis of data, provision of advice, or similar services. Persons performing such ancillary services may be recognized in the Acknowledgments section.

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Type on one side of white bond paper, 216 × 279 mm (8 in. × 11 in.) with margins of at least 25 mm (1 in.) on all sides of the page. ***Double-space*** the entire manuscript (three lines per vertical inch). Number all pages in upper-right corners. Indent paragraphs 5 spaces. Do ***not justify***. Do ***not*** underline titles, headings, or other words. Do ***not*** type authors' names or other identification anywhere except on the title page. Repeat title only (no authors) on the abstract page. Begin each of the following on a new page: title page, abstract, text, product-sources list, acknowledgments, reference list, each table, each appendix, list of figure legends. Use standard English. Employ the first person and active voice (eg, "We believe that pigs can fly") rather than the 'obscure person' and passive voice (eg, "It is believed that pigs can fly")—because the latter obscures the identity of the responsible party (the believer).

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Case Report: Title Page, Introduction, Case Summary, Discussion, References, Tables, Figure Legends.

Review Article: Title Page, Table of Contents, Introduction, Review of the Literature, State-of-the-Art Summary, Acknowledgments, References. Tables, appendices, and illustrations may be included. Other formats may be suitable.

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Introduction: Briefly describe the background of the work or the paper. Cite only pertinent references, and do not review the subject extensively. Do not include data or conclusions from the work reported in your paper. In a research paper, end this section with a clear statement of the research question(s) or hypothesis(es).

Methods Section (in a research paper): Describe the selection of patients, controls, or laboratory animals. Give details about randomization. Describe methods for blinding of observations. Give numbers of observations. Report losses to observation (eg, dropouts or disqualified subjects), listing numbers of subjects or data sets lost, when lost, and why lost. *Describe methods in sufficient detail to allow other workers to replicate your work.* Give references to established methods; provide references and brief descriptions for methods that have been published but are not well known; describe new or substantially modified methods, give reasons for using them, and evaluate their limitations. Report calibration of measuring devices.

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brand names may be given in parentheses after generic names. Commercial Products—Identify any commercial product (including model number if applicable) the first time it is mentioned, giving the manufacturer's name, city, and state or country—in parentheses in the text. If four or more products are mentioned, do not list any manufacturers in the text; instead, list them on a Product Sources page at the end of the text before the References. Provide model numbers when available and manufacturer's suggested price if the study has cost implications.

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Results Section: Present results in logical sequence in the text. Tables and illustrations may also present data. Do not repeat in the text all the data in the tables or illustrations; emphasize or summarize only important observations and trends. Be sure to report all the results; do not save some of them for the Discussion section. Do not discuss the findings in the Results section. Exact p values are preferred in all cases but are *essential* when values are not statistically significant. Do not report original results merely as nonsignificant or NS.

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Code Blue, Vital Signs Inc, Totowa NJ, $19.85

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Acknowledgments Page: On this page you may recognize the services of persons who made ancillary contributions to the work or the manuscript. Such services might be advice about methodology; data collection; statistical advice or analysis; equipment selection or operation; cooperation as caregiver, patient, or subject; manuscript preparation; in-house review; and other services. Each acknowledgment must specify the service rendered. Named persons must provide written agreement (accompanying submitted manuscript) to be so recognized.

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Use of References: References are used to support statements of fact, to indicate sources of information, or to guide readers to further information. Be careful to make clear in the text the reason for a specific citation (ie, do not imply support of a statement of fact by citing a reference that simply addresses the issue). Cite only sources that have actually been consulted and evaluated by the authors. Cite only ***published or accepted*** material. Cite original articles in preference to textbooks, review articles, abstracts, editorials, or letters. Avoid citing abstracts because some published abstracts have never been subjected to peer review and may not support your contention. Make every effort to determine whether an abstract has been subsequently published as a full-length paper. Avoid citing non-English language sources. When citing from a book, specify the page numbers unless you are citing the entire book. If you cite a paper that has been accepted but not yet published ("in press"), provide a copy of the paper to the Editor when you submit your manuscript.

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Article in a journal carrying pagination throughout volume:

1. Shepherd KE, Johnson DE. Bronchodilator testing: an analysis of paradoxical responses. Respir Care 1988; 33:667-671.

Article in publication that numbers every issue beginning with Page 1:

2. Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

3. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33:1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to allow retrieval.)

4. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3, Suppl):139s-143s.

Abstract in journal: (Abstracts are not strong references. Abstracts more than 3 years old should not be cited. When cited, abstracts should be identified as such.)

5. Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35:1087-1088.

Editorial in journal:

6. Rochester DF. Does respiratory muscle rest relieve fatigue or incipient fatigue? (editorial). Am Rev Respir Dis 1988;138:516-517.

Editorial with no author given:

7. High frequency ventilation (editorial). Lancet 1991; 1:706-708.

Letter in journal:

8. Smith DE, Herd D, Gazzard BG. Reversible bronchoconstriction with nebulised pentamidine (letter). Lancet 1988;2:905.

Paper accepted but not yet published:

9. Hess D. New therapies for asthma. Respir Care (year, in press).

Personal author book: (Specific pages should be cited whenever possible.)

10. Nunn JF. Applied respiratory physiology. New York: Appleton-Century Crofts, 1969.

Note: To specify pages in a book, place a colon after the year and then list the page(s). Examples: 1969:85 (one page), 1969:85-95 (series of contiguous pages), 1969:85,95 (separated pages).

Corporate author book: (Specific pages should be cited whenever possible.)

11. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group, 1977.

Book with editor(s): (Specific pages should be cited whenever possible.)

12. Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott, 1977.

Chapter in book: (Specific pages should be cited whenever possible.)

13. Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Phildelphia: JB Lippincott, 1977:171-223.

Newspaper article:

14. Rensberger B, Specter B. CFCs may be destroyed by natural process. The Washington Post 1988 Aug 7:Sect A:2(Col 5).

Dictionary or similar reference:

15. Pneumohemopericardium. Dorland's illustrated medical dictionary, 26th ed. Philadelphia: WB Saunders, 1981: 1038.

Tables: Use tables to display information, compare data, or show trends. Start each table on a separate page. Do not construct a table with fewer than two lines (rows) or columns of data (instead, put the data in the text). Avoid more than 8 col-

umns across. Number tables as Table 1, Table 2, etc, consecutively in the order of their first mention in the text. Place the number and a ***descriptive*** title above the table (not on a separate page). Give each column a brief heading. Place explanatory matter in footnotes, not in the title or column headings. Explain in footnotes all nonstandard abbreviations and symbols used in the table. To key footnotes to the table body, use conventional designations (asterisk, dagger, double dagger, etc) in consistent order, placing them superscript in the table body.

Double-space all elements of tables, including titles, column headings, data, and footnotes. Continue a deep table on following pages. Do not use horizontal or vertical rules. Do not submit tables as photographs, or reduced in size, or on oversize paper. Use the same typeface as in the text. Supply the name and version of any table-building computer program used.

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Authors in This Issue

Becker, Ellen 153
Branson, Richard D 138
Dedman, Shelley 147
Durbin, Charles G Jr 123
Farias, Emidio M 154
González-Lawless, C Laura 147
Hess, Dean 105
Heulitt, Mark J 147
Lawrence, Gretchen 148
Marley, Rex A 149
Martin, D Wayne 152
Millard, Mark W 148
Rendell-Baker, Leslie 154
Sabo, John S 147
Suzor, Dixie 153

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